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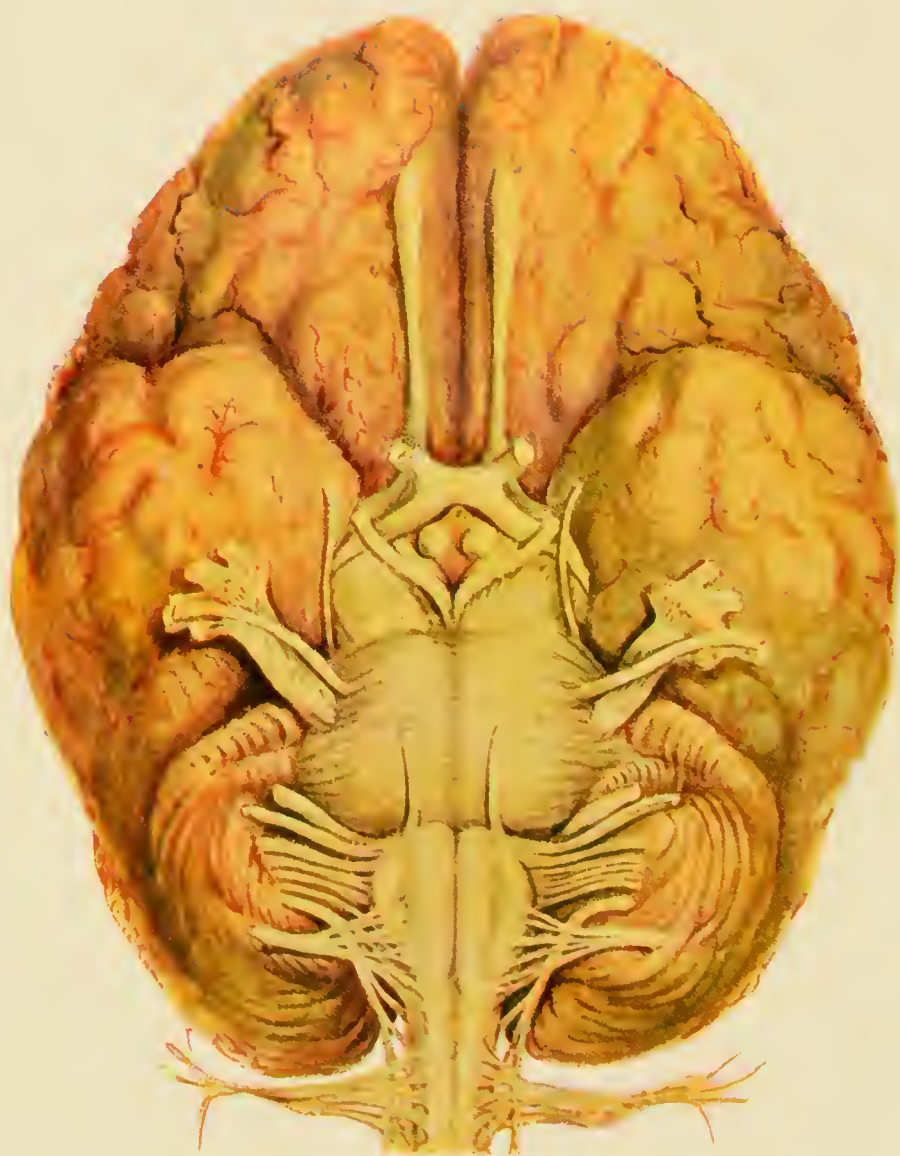
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THE BASE OF THE BRAIN.

(From a Painting by Sir Charles Bell at the Middlesex Hospital Medical School.)

DISEASES OF THE NERVOUS SYSTEM

BY

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WITH 8 COLOURED AND 12 BLACK-AND-WHITE
PLATES, AND 101 FIGURES IN THE TEXT

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To

JAMES KINGSTON FOWLER, M.D.

IN GRATEFUL APPRECIATION OF HIS
TEACHING AND COUNSEL

PREFACE

IN attempting to marshal the main facts of neurology in a concise and yet, I hope, readable manner, it has been my constant aim to bear in mind the principal difficulties which are apt to beset the student.

The experience on which this book is founded has been obtained for the most part at the Hospital for Nervous Diseases, Maida Vale, and at the clinics for nervous diseases which I have for some years past held at the Middlesex Hospital. To my colleagues at both those hospitals I express my hearty thanks for the many opportunities they have kindly given me of observing cases under their care.

To the courtesy and generosity of Professor Pierre Marie in supplying me with photographs for illustrations, I owe a special debt of gratitude, as also I do to Mr. Bland-Sutton for the loan of blocks, and to others who have kindly allowed me to reproduce illustrations, the sources of all of which, I trust, are duly acknowledged.

For taking photographs of my cases I have

chiefly to thank Mr. Mann, and for the taking of the instantaneous photographs reproduced in four of the plates I am indebted to Mr. Charles Urban.

Finally, my best thanks are due to the publishers for the valuable assistance they have given me throughout the preparation of the book.

H. CAMPBELL THOMSON

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DISEASES OF THE NERVOUS SYSTEM

SECTION I.—INTRODUCTORY

CHAPTER I

GENERAL STRUCTURE OF THE NERVOUS SYSTEM

THE nervous system is composed of nerve cells surrounded and supported by connective tissue. Each nerve cell consists of a cell body and its processes. The processes which conduct impulses away from the cell body are called axons, while those which transmit impulses towards the cell body are known as dendrons, and the cell body together with all its branches constitutes a **neuron** (Fig. 1). The neuron is therefore a cell which has become specially differentiated to perform the duty of conduction.

As a general rule, the axon is single and of greater length than the dendrons, and, as already mentioned, it conveys impulses away from the cell body and gives off branches (known as collaterals) along its course. Shortly after leaving the cell body the axon becomes encased successively with a sheath of myelin and a neurilemma or sheath of Schwann, both of which it discards a little before it breaks up into its terminal branches.

Such is a typical neuron, but, of course, the size and shape of the cell bodies and the length and thickness of their branches vary greatly, and the axon is not necessarily in all cases the longest branch.

It thus comes about that the nervous system is made up of an infinite number of neurons, bound

up by connective tissue into various bundles and tracts according to the different kinds of impulses they conduct. There are, for example, special bundles for motor impulses—the motor tract—and others for sensory impulses—the sensory tract—and so on, but the ultimate elements making up all these bundles are simple neurons.

It is generally thought that every neuron is a separate anatomical unit capable of living and dying independently of every other neuron, and that the processes of one have no physiological connection with those of another, *i.e.*, the branches are contiguous but not continuous, just as the branches of trees, however intimately entwined with those of others, depend for their vitality upon the integrity of their own stems and roots.

Doubt has occasionally been cast upon the correctness of the view that every neuron has a complete individuality, but the distribution of

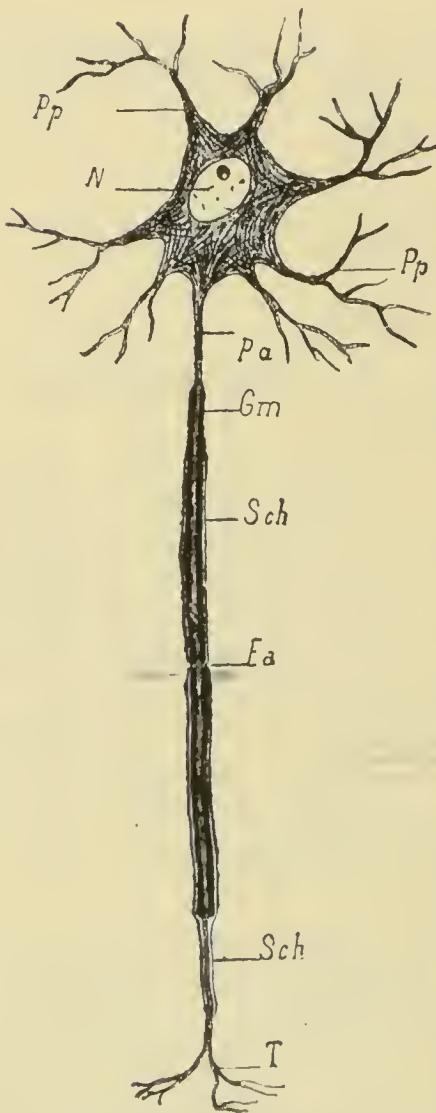


Fig. 1.—Diagrammatic representation of a neuron. (Morat.)

N, nucleus of the nerve cell; Pp, protoplasmic prolongations; Pa, axis cylinder; Gm, sheath of myelin; Sch, sheath of Schwann; Fa, annular node; T, terminal ramifications of the axis cylinder.

correctness of the view that every neuron has a complete individuality, but the distribution of

nerve degeneration, which is mainly limited by the termination of cell branches of particular tracts, and the embryological evidence of the independent development of different units, are both strongly in favour of its truth.

A clear conception of the constitution of the nervous system as made up of collections of neurons is an important aid to the understanding of all nervous diseases, for it enables us to foretell to a large extent the course and limits of degenerations and to understand why certain tracts are affected to the exclusion of others.

THE MOTOR NEURONS

The neuron theory of the constitution of the nervous system makes it easy to understand the mechanism by which motor disabilities are produced, and at the same time supplies a guide to the symptoms that are likely to arise from lesions at different levels of the tract.

The path by which motor impulses are transmitted consists of two neurons, upper and lower (Plate I.).

The upper neuron has its cell body in the motor area of the cerebral cortex, and while its short branches interlace with others in all directions, its long one conducts impulses down to the spinal cord, and, with countless others of similar structure, helps to make up the motor tract. After leaving the cerebral cortex these fibres pass successively through the corona radiata, internal capsule, crus cerebri, pons, and medulla, where the majority of them decussate, and then travel down the crossed pyramidal tracts of the spinal cord to terminate finally alongside the corresponding branches of the cells of the anterior horns.*

Those fibres which do not decussate in the medulla travel down the cord by the direct

* Probably the axis-cylinders of the upper neurons synapse with the cells of Clarke's column, and thus their communication with the cells of the anterior horn is only indirect.

Cerebral cortex
1

Corona Radiata

Int. Capsule

Crus Cerebri

pons

Medulla.

X

decussation

Crossed pyramidal

2

Direct Pyramidal

pyramidal tracts, and ultimately cross to the other side.

It has generally been considered that the fibres of the direct pyramidal tract cannot be traced much below the mid-thoracic region, but Risien Russell* has shown that they can be followed into the sacral segments of the cord, and that decussation on their way down the cord takes place in the anterior commissure.

The **lower neuron** has its origin in the cells of the anterior horn, from which the axon conducts the motor impulses to the muscles.

Since every impulse giving rise to a voluntary movement must of necessity pass down these paths—for there are no alternative routes—it follows that an interruption of either of these neurons will be accompanied by loss of voluntary movement; or, to regard the matter in its clinical aspect, a patient complaining of loss of voluntary power must, except in the case of functional disorders, have a lesion somewhere in the motor paths which prevents the impulses from passing

The next step is to consider what differences exist between an interruption of the fibres of the upper and of the lower of these two neurons; and here there is not, as a rule, much difficulty, for the impulses which travel down the upper neuron, besides giving rise to voluntary movements, also exercise restraint over the actions of the lower neuron, so that when this restraint is removed there is found, in addition to loss of power, some rigidity of muscles and increase in the deep reflexes of the affected parts. But, inasmuch as the lower neuron is independent of the upper as far as its actual existence is concerned, there is no wasting of muscles except such as occurs from disuse, neither is there any change in their electrical reactions. On the other hand, if the lower neuron is injured there is not only loss of power, but also a loss of

* *Brain*, Part lxxxii., 1898.

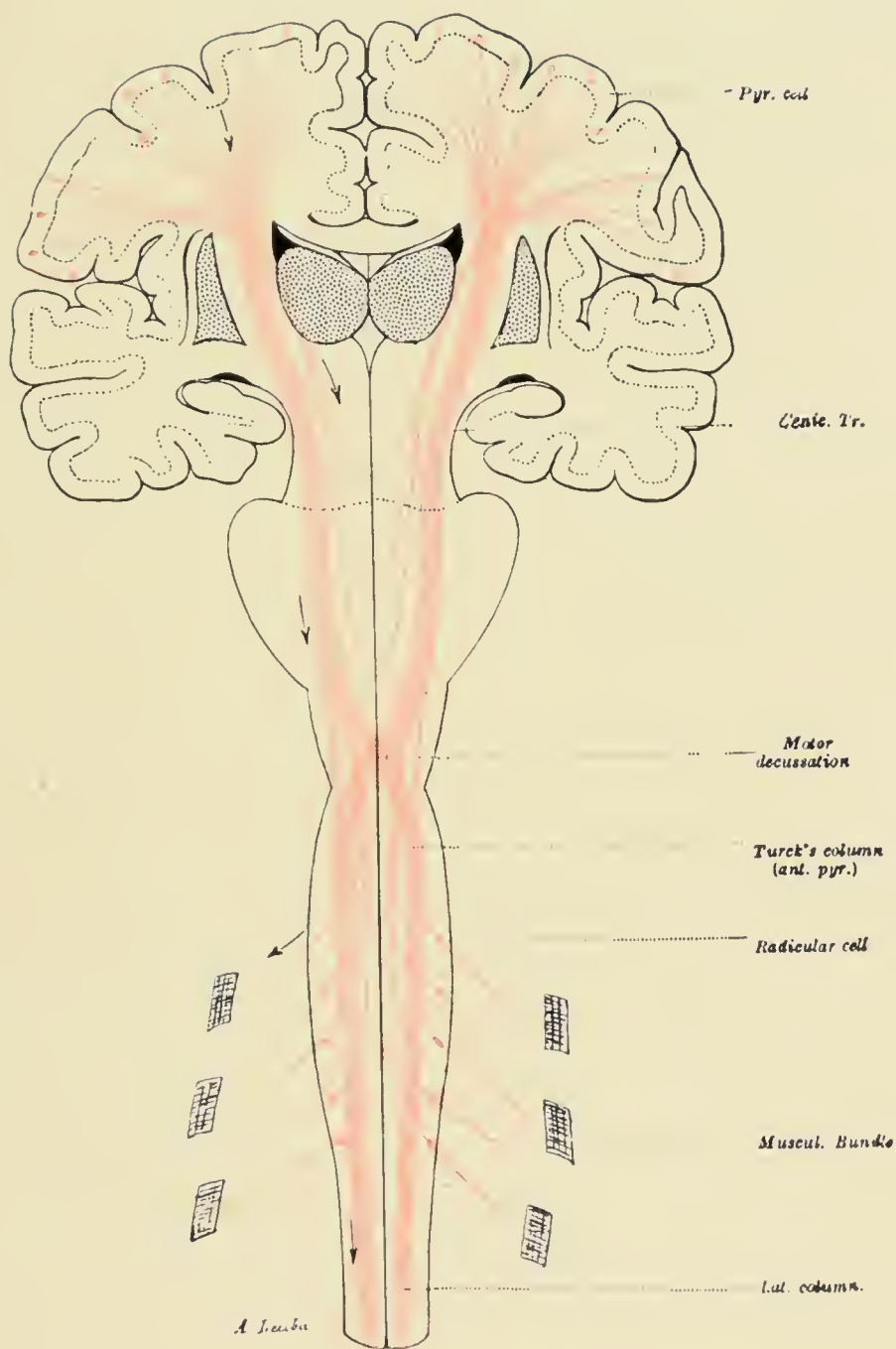


PLATE 1 - Motor Field with its Two Principal Orders of
Fibres of Projection. (Morat.)

reflexes (if any happen to be situated in the affected zone), together with wasting, flaccidity, and changes in the electrical reactions of the muscles, for the nutrition of the muscles is directly dependent on the vitality of the lower segment.

These differences may be conveniently summarised in the following table:—

	LESION OF UPPER NEURON.	LESION OF LOWER NEURON.
1. Wasting . .	Slight, and only in consequence of disuse	Wasting a prominent feature.
2. Reflexes . .	Deep reflexes increased; plantar reflex gives an "extensor response"	Abolished.
3. Rigidity . .	Limbs tend to become rigid	Limbs tend to become flaccid.
4. Electrical reactions	No obvious changes . .	Reactions to faradism and galvanism modified. Frequently a typical "reaction of degeneration."
5. Contractures	Rigidity accompanied by contractures in which flexion and adduction overcome extension and abduction	Irregular deformities owing largely to unopposed actions of non-paralysed muscles.

It will be seen that increased reflexes and rigidity tend to go together, while flaccidity, wasting, and loss of reflexes generally accompany one another.

The importance of this table to the student is that in it he has the basis of all the symptoms which depend upon loss of power, and instead of endeavouring to remember a different set of points for every individual instance he can apply the general principles to his case and then vary the particulars as required.

For instance, the motor symptoms underlying primary or secondary lateral sclerosis and ataxic paraplegia will all, in so far as they depend upon

a lesion of the upper neuron, obey the general rules of such lesions. Modifications will merely concern methods of onset, course, and such like details. On the other hand, infantile paralysis, progressive muscular atrophy, and peripheral neuritis have their symptoms all founded on a lower neuron lesion.

Sometimes the two conditions are mixed, as in amyotrophic lateral sclerosis, where the usual clinical picture is that depending on a lesion of the lower neuron as far as the hands are concerned, and upon one of the upper neuron as regards the legs, which signifies that the cells of the anterior horns in the cervical region of the cord are degenerating at the same time as those fibres of the crossed pyramidal tracts which carry voluntary impulses to the legs.

THE SENSORY NEURONS (Plate II.)

The neurons for the conduction of sensations are more numerous and arranged in a more complicated manner than those for movements. It is necessary to consider their arrangement and the effects produced by their injury or disease (1) in the peripheral nerves, (2) in the posterior roots, (3) in the spinal cord, and (4) in the brain.

1. SENSORY NEURONS IN THE PERIPHERAL NERVES

Head, Rivers, and Sherren, and Head and Sherren, have shown that at the periphery the afferent nerves can be divided into three systems—(1) the system for deep sensibility, (2) the protopathic system, (3) the epicritic system.

In (1) the system for deep sensibility sensations arising from pressure and from movements of tendons, joints, and muscles are appreciated.

The locality of the stimulus and the direction of movement in a joint can be appreciated, as also can pain if the pressure is excessive or if a joint is injured. The fibres which conduct the impulses

for this form of sensibility run chiefly with the nerves to the muscles, and they are not destroyed by dividing all the sensory nerves to the skin.

Thus, when the sensation to light touch is entirely lost in the skin, a sense of pressure and of movements of joints may still be appreciated provided the deep system is intact.

In (2) the **protopathic system** the fibres respond to painful cutaneous impressions and to the extremes of heat and cold.

In (3) the **epicritic system** accurate cutaneous localisation of light touch, the discrimination of the points of a compass, and the recognition of the slighter differences in temperature are accomplished.

A knowledge of the existence of these three systems is obviously of the greatest importance in practical medicine.

In the first place it shows what care it is necessary to take in examining for sensibility, for when cutaneous nerves are severed an appreciation of slight pressure through the deep system may easily lead, as it often has in the past, to an erroneous supposition that there is no anæsthesia. In testing for the tactile sensibility of the skin the very lightest form of stimulation, such as can be obtained with cotton-wool, is required.

Moreover, the lost protopathic and epicritic systems of sensibility are not always equal in extent or intensity. When, for instance, union is taking place between the ends of a divided nerve the sensation to painful stimuli returns first. For a time the pain cannot be localised, but radiates widely from the spot stimulated, though always in definite directions.

Together with return of sensibility to pain there is also a return of appreciation of extremes of temperature, *e.g.*, great heat or great cold, which Head has shown to be due to the presence of "heat" and "cold" spots. With the return of

sensations for pain the nutrition of the skin also improves, and any ulcers that may have been present readily heal. Gradually the skin over the affected areas again becomes sensitive to light touch and to slight differences of temperature.

It therefore appears that the appreciation of touch and pain is subserved by independent fibres, since Head has been able to demonstrate the dissociation of these two forms of sensation, and also that fibres which conduct the painful impressions are capable of regenerating more quickly than those which conduct the tactile impulses. Head has summarised the characteristics of the three systems in the following table:—*

Loss of **epicritic sensibility** abolishes—

Recognition of light touch over hairless parts or parts that have been shaved.

Cutaneous localisation.

Discrimination of compass points.

Appreciation of differences of size, including the accurate discrimination of the head from the point of a pin apart from the pain of the prick (acuæsthesia).

Discrimination of intermediate degrees of temperature from about 25° C. to about 40° C.

Loss of **protopathic sensibility** abolishes—

Cutaneous pain, especially that produced by pricking, burning, or freezing, together with that of stimulation with a painful interrupted current. Over hair-clad parts, plucking the hairs ceases to be painful.

Sensations of heat from temperatures higher than about 45° C.

Sensations of cold from temperatures below 20° C.

After destruction of all cutaneous afferent fibres the part is still endowed with deep sensibility.

* *Brain*, Part cxvi., 1906.

Pressure can be recognised, and its gradual increase appreciated. Pain is produced by excessive pressure (measured by the algometer). Movements of muscles can be recognised.

The point of application of pressure can be localised.

The patient can recognise the extent and direction of movement produced passively in all the joints within the affected area.

Muscle sense.—Sensations from muscles, joints, and ligaments are generally grouped together under the term “muscle sense,” and their loss, singly or combined, is largely responsible for the incoordination in locomotor ataxy.

The sense of the muscles can be tested by finding whether the patient can distinguish the differences in weight of objects of similar size, and allied to this is the power of recognising with accuracy the shapes of objects, *i.e.*, the stereognostic sense. ||

When the sensibility of the joints is impaired the patient cannot tell in what position his limbs are placed, and this can be demonstrated by putting the limbs in various positions and requesting him (while his eyes are closed) to describe them or to put his other limbs into similar positions.

The sensibility of the bones can be tested by means of vibrations produced by placing a tuning-fork on them. This sensation, like those from the muscles and joints, is frequently impaired in locomotor ataxy.

2. SENSORY NEURONS IN THE POSTERIOR ROOTS

The distribution of anæsthesia in root lesions differs from that of the peripheral nerves inasmuch as the fibres are so arranged that each root corresponds to a definite segment of skin. The segmentary distribution of the posterior roots has been investigated by many observers, and with exceptional thoroughness by Head, as illustrated by the accompanying diagrams (Figs. 2 and 3), from the

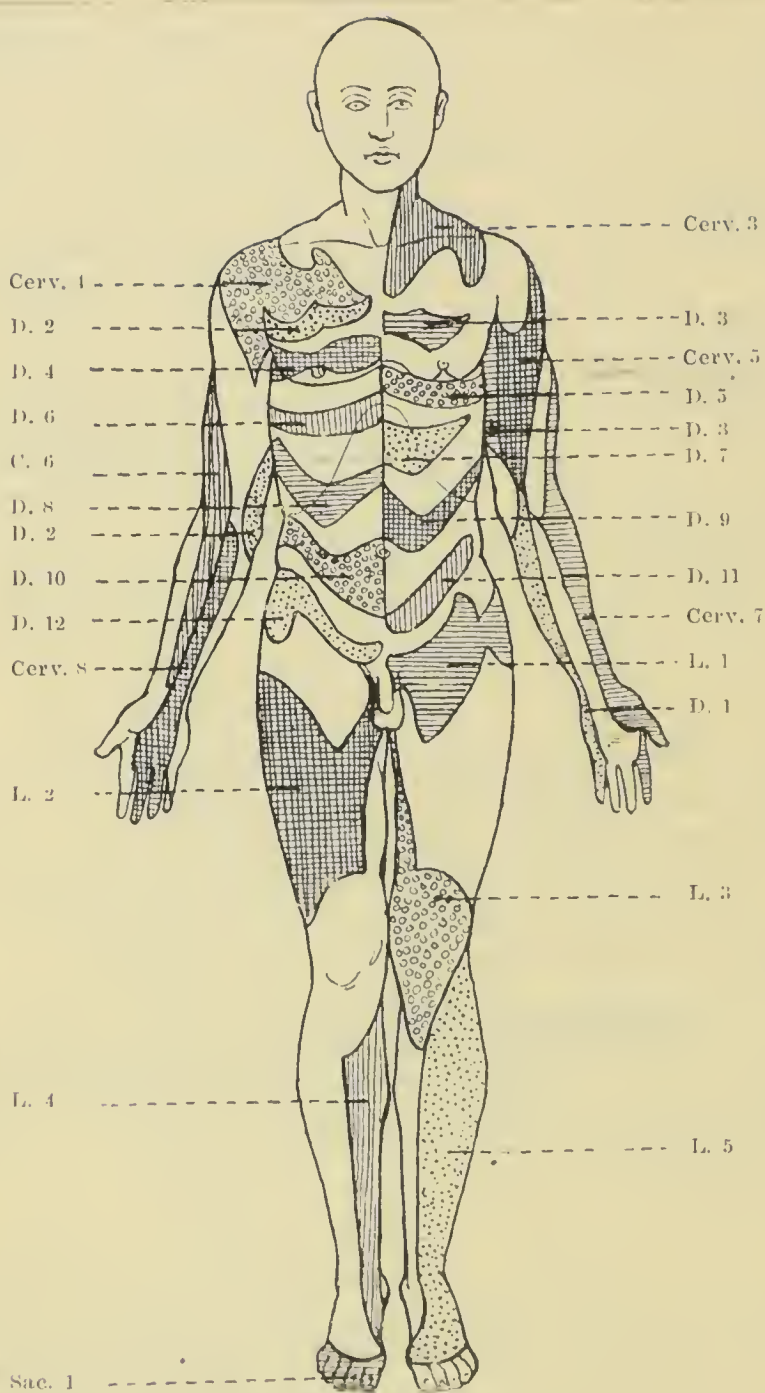


Fig. 2.—Sensory segmental functions of the spinal cord. (*Heal.*)

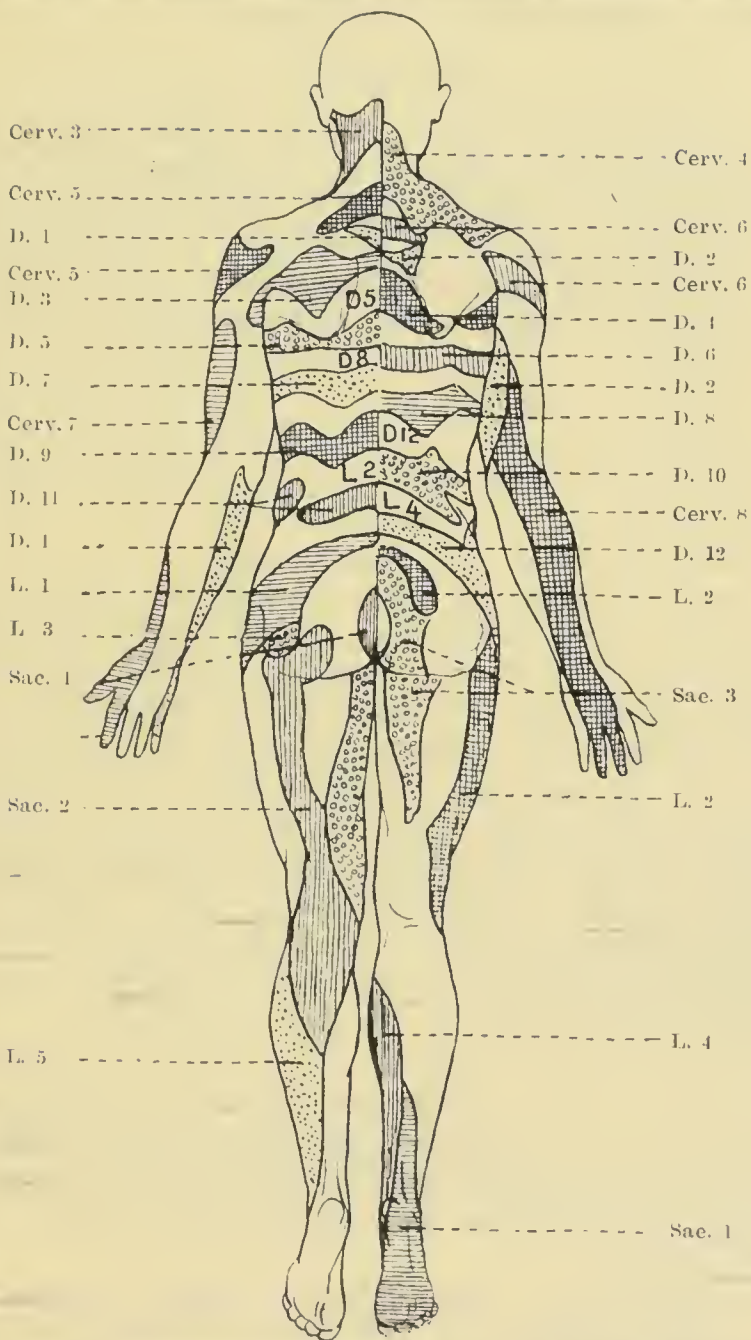


Fig. 3.—Sensory segmental functions of the spinal cord. (*Head.*)

areas occupied by herpes zoster, which is due to lesions of the posterior root ganglia.

3. SENSORY NEURONS IN THE SPINAL CORD

The principal sensory tracts in the cord are: (1) the posterior columns, (2) the direct cerebellar tracts, (3) the antero-lateral tracts of Gowers, and (4) the ascending tracts in the anterior columns.

(1) The posterior columns.—The posterior columns are divided into posterior external (column of Burdach) and posterior internal (column of Goll). The fibres in these columns, with the exception of a small number which pass between various segments of the cord (endogenous), are a direct continuation of those in the posterior roots, and are known as exogenous fibres.

(a) A number of these exogenous fibres pass directly up to the top of the same side of the cord and terminate in the nucleus gracilis and the nucleus cuneatus. On their way up these branches give off numerous collaterals which terminate in the cells of the grey matter. From the nucleus gracilis and nucleus cuneatus there starts a second relay of fibres which cross and terminate in the optic thalamus of the opposite side. From the optic thalamus springs a third set which spreads out and terminates in the cerebral cortex.

The chief function of these long fibres of the posterior columns appears to be that of conducting impulses for tactile discrimination (i.e., compass test) and for passive movements and position, since these sensations are not lost on the opposite side to a Brown-Séquard lesion of one half of the cord (Head).

Destruction of these columns usually gives rise to ataxy, though the influence in this direction of a possibly associated lesion of fibres connected with the direct cerebellar tract must not be lost sight of.

(b) There are fibres which run a variable dis-

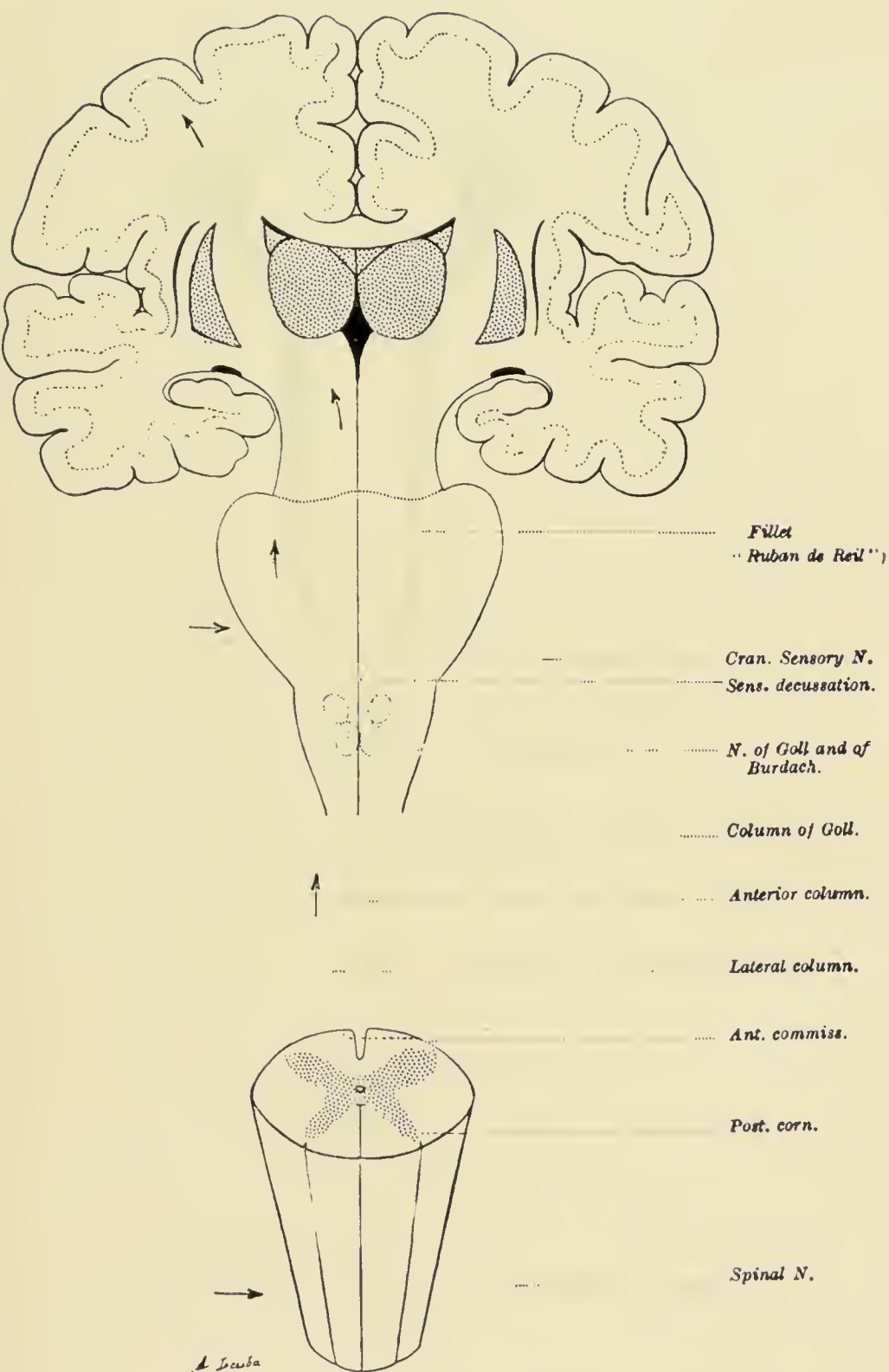


PLATE II.—Sensory Field with its Two Principal Orders of Fibres of Projection. (Morat.)

taunce up the cord and then cross. Their function also seems mainly to be that of conducting tactile impulses, and, owing to the distance they run in the cord before crossing to the ascending columns of the opposite side, they too are often apt to escape on the opposite side to the lesion in a Brown-Séquard paralysis (Head).

(c) There are fibres which mainly conduct impulses for pain. These probably terminate very soon after they reach the cord in cells of the grey matter from which a second set of fibres arise and cross to the opposite side and ascend in the antero-lateral tracts. The course of the impulses for heat and cold seems to be practically the same as for those of pain.

(2) **The direct cerebellar tract.**—Some of the fibres of the posterior roots terminate in a group of cells, known as Clarke's column, which are situated at the base of the posterior horns. From Clarke's cells there arise a set of neurons which form the direct or dorso-lateral cerebellar tract and which terminate in the cerebellum.

These fibres conduct impulses of co-ordination from bones, joints, ligaments, and muscles, and injury to them is followed by ataxy.

(3) **Antero-lateral tract.**—This tract is composed of fibres which arise from cells in the grey matter of the cord, and of which (1) some pass upwards to the cerebellum chiefly by way of the superior peduncle, while (2) others terminate in the optic thalamus of the same side.

As already stated, the antero-lateral tract serves as the main path for impulses of pain and temperature sensations.

(4) **Ascending tract in anterior column.**—In the anterior column there are some ascending fibres which come from the opposite side of the cord and eventually terminate in the optic thalamus. This tract appears to be one of the main paths for those impulses of tactile sensibility which

have crossed from the other side of the cord and are making their way upwards.

Path of impulses in the cord.—It has been recently shown by Henry Head and Theodore Thompson* that the grouping of the sensory impulses in the cord is quite different from that of the peripheral nerves in which they run in three main streams by the epicritic, protopathic, and deep sensibility fibres. In the cord the impulses for the different forms of sensation tend to unite, irrespectively of the various paths by which they have arrived. Thus the tactile, painful, and thermal sensations are all collected together in their own groups, and the thermal sensations are further particularised into those for heat and cold, so that in some cases the sensations for one may be lost while those for the other are retained. This new grouping appears to take place on the same side of the cord as that by which the impulses enter, but after the new formation has taken place those of tactile sensibility, tactile localisation, painful and thermal sensibility, cross sooner or later to the other side. But those which are concerned with tactile discrimination,† i.e., recognition of two points of a compass, and those of passive position and movement, pass straight up the posterior columns to cross ultimately higher up.

The main types of *cord anaesthesia* that can be recognised are those arising—

- (1) From transverse lesions.
- (2) From lesions of the grey matter.
- (3) From lesions of one half of the cord.

In lesions which extend across the cord, all forms of sensibility will be affected below the injury. If the lesion is complete there will be total

* *Brain*, Part cxvi.

† It is important to notice that Head uses the compass method to test the power of tactile *discrimination* and not for tactile *sensibility*; and although loss of tactile sensibility will affect the power of tactile discrimination it is possible to find loss of discrimination when tactile sensibility remains normal.

anæsthesia below its level, with probably a zone of hyperæsthesia just above, owing to irritation and inflammation of the nerve structures in the immediate vicinity of the injury.

With disease of the commissural grey matter there is very apt to be loss of sensation of pain and temperature, while that of touch is preserved—a dissociation particularly well seen in cases of syringomyelia.

When injury or disease is sharply limited to one half of the cord, as may occur in stab or bullet wounds, the symptoms known as Brown-Séquard paralysis are liable to follow: on the side of the lesion and below its level, loss of power, loss of sense of position, of tactile discrimination* (Head), and of vasomotor tone, while on the opposite side there is loss of sensation to touch (partly), and loss of pain and temperature senses. Head has shown that while the loss of pain and temperature senses is constant in these cases, those of touch and pressure are more rarely affected, due, presumably, to their crossing less rapidly than the others.

At the level of the lesion, and on the same side, there is a zone of anæsthesia corresponding to the area supplied by those posterior roots which the lesion actually destroys, and just above the level of the lesion there is some hyperæsthesia due to irritation.

Referred pains.—In diseases of the various viscera, the pain to which they give rise is frequently “referred” to a part of the body which seemingly has no direct connection with the seat of the disease. Thus the headache from eye-strain may be situated over the forehead, and pain between the shoulders is a common accompaniment of gastric disorders.

The distribution of referred pains has been especially investigated by Head, who has con-

* *I.e.*, the power to respond to the compass test.

structed a scheme of intramedullary segmentation based on the hypersensitive areas which result from irritation of viscera.

The explanation is that the impulses which travel up to the cord from the diseased organs by the sympathetic fibres are reflected outwards along the sensory roots of the segment at which they happen to enter, and so the patient often refers the pain to the area of distribution of these roots rather than to the viscus from which the abnormal impulses originally arise.

4. SENSORY NEURONS IN THE BRAIN

At the **optic thalamus** the fibres which carry sensations from the opposite side of the body are collected together, and disease of the thalamus or in its vicinity is very apt to be accompanied by anæsthesia over the opposite half of the body. This may be the case in tumours of the thalamus or in lesions of the posterior third of the posterior limb of the internal capsule through which the fibres pass before radiating to reach the cortex.

In the cortex. — There is still some uncertainty concerning the precise distribution of sensory fibres in the cortex. The fact that lesions of the “motor area” are frequently associated with some loss of sensation has led to the view that sensation and movement are both represented in the motor area. The correctness of this conclusion requires to be examined in the light of Sherrington and Grünbaum’s work, which shows that the motor area does not extend backward farther than the fissure of Rolando. The post-central convolution can therefore no longer be regarded as motor in function, but there is strong experimental and pathological evidence to show that it is the seat of sensory representation.

Mills* considers that the “cortical representation of cutaneous and muscular sensibility is in-

* *Journ. of Nervous and Mental Diseases*, Oct., 1906.

dependent of motor representation, that it surrounds the motor zone, and that it is subdivided into a mosaic of centres, each centre or group of centres being anatomically and functionally correlated to a motor centre or centres." He also considers that stereognostic representation has its independent cortical area, which is probably situated in the parietal lobe.

The type of sensory disturbance from lesions of the post-central convolution is mainly impairment of tactile sensibility and inability to localise with exactness the spot that is touched, a condition known as "atopognosis." Sir Victor Horsley* shows that in these circumstances the spot indicated by the patient is higher in the limb than that actually stimulated. Tactile sense is probably represented in the gyrus fornicatus also.

* *Brain*, 1906, p. 137.

Atopognosis .- impairment of tactile sensibility + inability to localise with exactness the spot that is touched.

CHAPTER II

THE REFLEXES

A REFLEX act consists of a contraction of a muscle or group of muscles in response to a sensory stimulus, and the elements necessary for its production comprise an afferent nerve, a "centre," and an efferent nerve, the whole being called a reflex arc.

In a simple spinal reflex the impulse travels along the sensory nerve to the spinal cord, which it enters through the posterior roots, and then, passing through the grey matter of the cord, reaches the motor cells of the anterior cornua, from which, in response to the stimulus, impulses pass out to the muscles and give rise to movements. Reflex acts can be carried out by a single segment of the cord independently of the higher centres, but they are modified by the latter, and may be voluntarily checked to a great extent.

Reflexes may be divided into various classes, *e.g.* :

Superficial reflexes, obtained by stimulating superficial nerves of the skin.

Deep reflexes, produced by striking certain muscles or tendons.

Visceral reflexes, as those of swallowing and micturition.

Bone reflexes, obtained by percussing various bony points in the upper and lower extremity.

The **principal superficial reflexes** are :

Plantar reflex, movement of foot and leg produced by stimulating sole of foot. S., 1, 2 and 3.

Gluteal reflex, contraction of gluteus on stimulating buttock. L., 4 and 5.

Cremasteric reflex, detraction of testicle on stimulating inner and upper aspect of thigh. L., 1 and 2.

Abdominal reflex, contraction of abdominal muscles on stimulating side of abdomen. D., 8, 9, 10, 11, 12.

Epigastric reflex, contraction of epigastrium on stimulating lower part of side of thorax. D., 4, 5, 6, 7.

Scapular reflex, contraction of scapular muscles on stimulating skin in region of scapula. C., 5, 6, 7, 8, and D., 1

Palate reflex, contraction of soft palate when touched.

Pharyngeal reflex, contraction on stimulating posterior wall of pharynx.

Conjunctival reflex, closure of eye on touching the conjunctiva.

The superficial anal reflex (3rd and 4th sacral) may be obtained by stimulating the skin of the perineum.

The bulbo-cavernous reflex, in which a contraction of the bulbous part of the urethra can be felt on stimulating the glans penis, depends on the 3rd and 4th sacral segments. This reflex is frequently lost in locomotor ataxy.

The case with which the superficial reflexes can be obtained in a healthy person varies, but from their reactions important inferences can often be drawn concerning (1) the fibres of the arc upon which their presence depends, and (2) the state of the upper motor segment, changes in which often modify the superficial reflexes.

The presence of a superficial reflex indicates that there is no interruption to the impulses passing through its arc, and thus affords information which is often useful for purposes of localising the level of the disease of the cord. Loss of the

reflexes, however, does not necessarily mean that their arc is broken, for some of them are at times abolished in diseases of the upper motor neurons. This is particularly the case with the abdominal reflexes, which, for instance, are apt to disappear in disseminated sclerosis and on the affected side in hemiplegia. There seems indeed to be a tendency for these superficial abdominal reflexes to be either lost or diminished by the same conditions that cause the deep tendon reflexes of the lower limbs to be increased.

THE PLANTAR REFLEX

The plantar reflex comprises those movements of the toes, foot, and leg which take place when the sole of the foot is stimulated.

Attention should be directed particularly to the big toe, which normally moves downwards, thus giving what is known as the "flexor response,"* but when there is any organic affection of the pyramidal motor tracts this movement of flexion is usually replaced by one of extension, and the reflex obtained is then known as the "extensor response," or Babinski's sign (Fig. 4).

Sometimes the plantar reflex is altogether absent, and in certain circumstances such absence is in favour of functional disease, but the importance of not assigning the same value to the absence of a symptom as to its presence is nowhere better exemplified, for the absence of the reflex is often but temporary, and a future examination may perhaps show a typical extensor response.

The best method of eliciting the reflex is to draw the thumb-nail, the head of a pin, or some similar object rather firmly along the sole of the foot; in some cases the outer, and in others the inner border of the sole gives the best results.

* Infants are an exception to this rule, for in them an extensor response is normally obtained up to about the age of twelve months, after which it is succeeded by a flexor response which persists so long as the motor tracts remain healthy.

Oppenheim has shown that the extensor response may also be obtained by stroking the inner side of the leg.

To constitute a satisfactory reaction, the big toe should move slowly and decisively up or down. If, as often happens, a brisk movement of the foot and leg leads to confusion, a lighter stimulation



Fig. 1.—Position of the big toe in the “extensor response”
(Babinski's sign).

will perhaps bring out the toe movement without that of the foot, or the movement of the latter may be controlled by the hand.

When the reflex is difficult to obtain or uncertain in direction, it is well to test it when the leg is loosely flexed and also when it is extended, for a reaction is sometimes better obtained in the one position and sometimes in the other. Damp

or cold feet make the reflex difficult to obtain, and may lead to an erroneous conclusion that it is absent.

Value in diagnosis.—A definite extensor response signifies the presence of some organic alteration of the pyramidal tracts, and it is therefore most useful as a test for distinguishing organic from functional disease.

Absence of all reflex movement whatever is suggestive of functional disease if other symptoms also point in this direction, but the same reliance cannot be placed on a negative as on a positive result, and therefore, while an extensor response indicates organic disease, neither flexor response nor absence of response necessarily excludes it.

Byrom Bramwell has noticed that in some cases of hemiplegia in which the extensor response is present on the paralysed side, stimulation of the sole of the foot on the non-paralysed side is not only accompanied by flexor response on that side, but also by a flexor response of the toe on the opposite, *i.e.*, the paralysed, side. To this reaction the term "crossed plantar reflex" has been given.

An extensor response is sometimes obtained for a little time immediately after an epileptic fit.

Among the cases in which the extensor response is of most value are undoubtedly those of early disseminated sclerosis, in which the patient complains of indefinite symptoms, combined, perhaps, with exaggerated knee-jerks and an abortive ankle clonus, all of which would still be compatible with a diagnosis of functional disturbance.

The principal deep reflexes are obtained by percussion of the tendo Achillis, of the tendons of the quadriceps extensor, and of the supinator longus and the triceps. The jaw reflex is obtained by sharply tapping the chin in a downward direction when the mouth is slightly open. These

reflexes are generally exaggerated when the pyramidal tracts above their level are diseased, while they are diminished or lost when the normal inhibitory influence of the pyramidal tracts is increased, and when the continuity of the reflex arc is interrupted.

THE KNEE-JERK

The knee-jerk is the most important of the tendon reflexes, and is obtained by sharply striking the tendon of the quadriceps extensor just below the patella, care being taken that all the muscles are relaxed.

In doubtful cases relaxation is best ensured if the patient clasps his hands together and pulls one against the other, his eyes being meanwhile raised towards the ceiling. This action, known as Jendrassik's method of reinforcement, occupies the attention and tends to remove any involuntary stiffening of the muscles.

Absence of the knee-jerk indicates organic disease, as also does inequality, but an increase may be found in functional as well as in organic disturbances.

The reflex arc for the knee-jerk is situated at the level of the 2nd, 3rd, and 4th lumbar roots, and anything which impairs the conductivity of the elements of this arc tends to diminish the reflex. Thus in peripheral neuritis, where the mixed nerve is at fault, the afferent impulses are blocked by the inflamed or degenerated peripheral sensory nerves, and even if they reach the cord the chances are that the motor fibres are unable to conduct them outwards. In locomotor ataxy the impulses are arrested at the posterior roots, while in infantile paralysis (of the lumbar region) the break occurs at the cells of the anterior horns. Impulses passing down the pyramidal tracts are constantly inhibiting the knee-jerks, so that to make the mechanism complete it is necessary to

consider these fibres in connection with those of the lower arc.*

An interruption in the continuity of the pyramidal fibres is accompanied by removal of the normal inhibition, and thus the knee-jerk is exaggerated, *e.g.*, in lateral sclerosis, transverse myelitis (above the lumbar region), and other diseases in which the pyramidal tracts are affected. On the other hand, irritation of the pyramidal fibres tends to increase their inhibitory influence, and diminishes the knee-jerk, as may be seen in some cases of meningitis and cerebral tumours.

To sum up: **The knee-jerks are lost—**

A. *From interruption of the reflex arc.*

(1) Injuries or disease of the lumbar cord causing destruction of the "centre."

(2) Diseases interrupting impulses to or from the lumbar region of the cord, *e.g.*, affections of anterior or posterior roots, mixed nerves, or muscles.

B. *From inhibition due to irritation of the pyramidal fibres, e.g., during early stages of cerebral hæmorrhage and fracture-dislocation of the spine, and in some stages of meningitis.* In many instances, *e.g.*, cerebral hæmorrhage, the irritation is only temporary, and the lost or diminished knee-jerk is soon succeeded by one that is exaggerated.

The knee-jerks are increased—

A. *From interruption in the conductivity of the pyramidal fibres above the lumbar region* whereby the normal inhibition is removed. This may be due to organic disease, *e.g.* :

(1) Traumatic, as in fracture-dislocation of the spine.

* Sometimes it happens that the peripheral ingoing and outgoing fibres are extraordinarily irritable for a short time before their conductivity is lost, and so the knee-jerks are occasionally found to be increased for a brief period during early stages of locomotor ataxy and peripheral neuritis. Such cases are, however, quite exceptional.

(2) Inflammatory, as in transverse myelitis.

(3) Degenerative, as in spastic paraplegia.

B. In functional disease. In many cases of neurasthenia and hysteria the knee-jerk is increased.

It will be observed that an exaggeration of the knee-jerk may accompany both functional and organic disease, but that absence of the knee-jerk always signifies the existence of an organic lesion.

There is one important *exception* to the rule that interruption of the conductivity of the pyramidal fibres above the lumbar enlargement is followed by exaggerated knee-jerks. When the spinal cord is completely divided, the reflexes below are lost, even though the division is far above their centres and has not apparently affected them. But for the reflexes to be lost in this way the transverse lesion of the cord must be complete; such, *e.g.*, may occur in cases of injury and compression from tumours. The importance of this loss in deciding as to the future of a patient with a fractured spine is obvious, for if, after sufficient time has elapsed for the initial shock to pass off, no knee-jerk can be obtained, it is highly suggestive and, taken with other evidence, often conclusive that the continuity of the cord has been completely severed.

There is no very satisfactory explanation of this exception to the rule. That suggested by Dr. Charlton Bastian,* by whom the persistent absence of deep reflexes after complete division of the cord was first noticed, supposes that in health the knee-jerk is constantly being influenced by impulses from the cerebellum as well as from the cerebrum. Those from the cerebellum are thought to have an accelerating influence, while, as we have already seen, those from the cerebrum, travelling by way of the crossed pyramidal tracts, have an inhibitory effect.

When the continuity of the lateral columns

* *Trans. Royal Med. Chir. Soc.*, 1890, p. 150.

is destroyed, the unantagonised cerebellar influence is, according to this idea, the cause of an

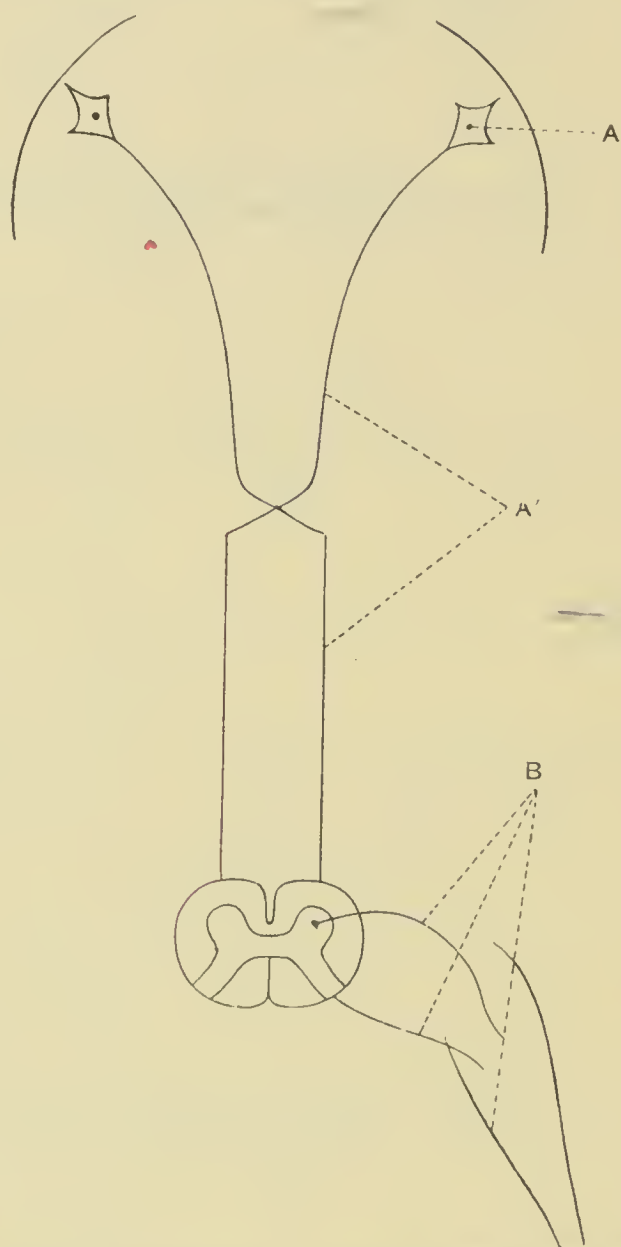


Fig. 5.—Diagram to show causes of main variations in the knee-jerk.

In lesions of the upper neuron (A and A') the knee-jerk is usually increased. In lesions of the lower neurons or of the quadriceps extensor muscles (B) the knee-jerk is usually lost.

increase in the knee-jerk, but when both cerebellar and cerebral influences are removed, as

they must be by a complete division of the cord, then the knee-jerks are permanently lost.

Assuming the existence of such cerebellar impulses, they must be widely diffused through the cord, since it takes a complete dissociation of continuity to block them.

Sir William Gowers has suggested a more simple explanation by supposing that the transverse lesion is followed by descending changes which cause such nutritional disturbance in the lumbar centres below as is sufficient to abolish their function without necessarily producing any recognisable change in their appearance.

The general rule concerning the increase of knee-jerks is, therefore, as follows: Interruption in the conductivity of the pyramidal fibres above the lumbar region is followed by exaggerated knee-jerks, except when the interruption is due to a complete transverse lesion of the spinal cord, in which case the knee-jerks are permanently lost.

The **chief visceral reflexes** to be considered are those of the pharynx, larynx, bladder, and rectum.

PHARYNGEAL AND LARYNGEAL REFLEXES

When these reflexes are impaired there is difficulty in swallowing and coughing. The food is apt to pass into the larynx without exciting cough, and the patient may be choked, or may suffer from broncho-pneumonia following irritation of the lungs.

BLADDER AND RECTAL REFLEXES

When the continuity of the spinal cord is interrupted above the lumbar enlargement the reflex arc for the bladder is cut off from all communication with the brain. Sensory impulses from the full bladder can no longer pass upwards, nor can impulses pass down to control the sphincter. The bladder, therefore, has to depend upon its own reflex, and it either fills and discharges

automatically, or, as frequently happens, the discharging reflex movement is not completely carried out, and the bladder becomes over-distended and finally overflows, producing "false" ineontinence. When the disease has destroyed the lumbar enlargement of the cord the reflex is lost; the walls of the bladder then lose their tone and cannot retain the urine, which continually runs away. Ineontinence may also occur from lesions of the cauda equina.

The usual result of a cord lesion upon the rectal sphincter is constipation and involuntary passage of fæces.

Rhythmical clonic contractions of certain muscles can often be obtained when there is a lesion of the upper neuron.

Ankle clonus is obtained by suddenly flexing the foot at the ankle-joint, when, on keeping up the pressure, the foot gives a series of jerks against the hand owing to the clonic contraction of the calf muscles. An ankle clonus does not exist in health, but is obtained when the pyramidal tracts are degenerated; hence it is generally associated with an increased knee-jerk and an extensor plantar response.

Sometimes, on trying to obtain ankle clonus, a few irregular contractions occur as the foot is being flexed, but cease when the pressure is increased and maintained. These are known as "spurious" ankle clonus, and do not in themselves indicate an organic lesion, although they do not, of course, negative the possible existence of one.

When the degeneration of the motor tracts is extensive, clonus can often be obtained in other situations, such as at the wrist, elbow, and jaw. A "patellar clonus" can also be sometimes elicited by suddenly pushing the patella in a downward direction, and so starting clonic contractions in the quadriceps extensor.

Flexors & Adductors >
> Extensors & Abductors.

CHAPTER III

RIGIDITY AND CONTRACTURES

WHEN the conductivity of the upper neurons is impaired there is a strong tendency for the muscles to become rigid.

The strength of the flexors and adductors overcomes that of the extensors and abductors, and the limbs gradually become fixed in position of flexion and adduction.

In the legs, particularly good illustrations of this tendency are to be seen in cases of infantile cerebral diplegia, in which the spasm of the adductors is sufficient to cause the legs to be crossed when walking is attempted. (Fig. 69, p. 270.)

In the arms, the result of rigidity is to be seen in hemiplegics, whose limbs tend to become flexed at the elbow, wrist, and fingers, and at the same time to be drawn close in to the side by the adductors.

This form of rigidity comes on slowly some time after the occurrence of an upper neuron lesion, and, when once established, is usually permanent. It is often spoken of as late rigidity, in contrast to the early rigidity which is apt to occur from irritation of the nerves soon after the onset of the disease. These late rigidities are referred to under the heading of Cerebral Hæmorrhage, in connection with which they are often prominent features.

The mechanism by which the rigidity that follows lesions of the upper neuron is produced

is not yet accurately known. Its occurrence has been ascribed by Dr. Hughlings Jackson to the unantagonised action of cerebellar influences, on the hypothesis that the cerebrum is the centre for changing movements and the cerebellum for continuous tonic movements, and that when the impulses from the cerebrum are cut off, those from the cerebellum come into undue predominance.

Another view is that the rigidity is due to chronic irritation of the cells of the anterior horns, induced by the degenerating motor fibres of the pyramidal tracts.

All that can be definitely stated at present is that permanent rigidity and contractures tend to follow lesions of the upper neuron.

In the later stages of rigidity, degenerative changes, followed by contraction, take place in the fibres of the muscles themselves and contribute further to permanent fixation of the joints.

Prevention of rigidity.—In all cases it is most important to try to prevent the onset of rigidity, which, when once it has set in, will effectually prevent any return of power that a partial recovery of the functions of the injured nerve fibres might otherwise allow.

Massage, electricity, and passive movements are the methods upon which reliance must be placed, and if these are to be of real use they must be practised with regularity and perseverance.

Massage maintains the vitality of the muscles and keeps them supple, the passive movements prevent the formation of adhesions round the joints, and electricity, judiciously applied, affords a further aid to both. Care must be taken to correct the tendency which the limb will show to remain in positions of flexion and adduction during the intervals of treatment, and, if necessary, it should be fixed in a favourable position.

especially during the night, by suitable splints or supports.

The masseur should pay particular attention to the nutrition and development of the weaker groups of muscles, viz., the abductors and extensors, so as to aid these as far as possible in resisting the greater power of the flexors and adductors.

Persistent treatment on these principles will do much towards averting the onset of rigidity, and will often make an immense amount of difference to the ultimate utility of a limb.

In lower neuron lesions also, *e.g.*, in the case of infantile paralysis, deformities are apt to arise from the fibrosis and subsequent contractions of the paralysed muscles, as well as from the un-antagonised action of the healthy muscles.

Here, again, a great deal of trouble may be obviated by massage, passive movements, and electrical treatment, care being taken to keep the limb in suitable positions during the intervals of active treatment. The contractions in this class of case can often be improved by some operation by which the tendons are cut or lengthened, but in most instances it is necessary also that the massage should be thoroughly carried out afterwards, otherwise but temporary benefit may be obtained from the operation.

Function of muscle substance.—Recent observations seem to show that muscle fibres are composed of two elements which differ from each other in their degree of excitability. It is thought that there are two contractile substances, one fibrillar and easily excited, and the other sarco-plasmic, of low contractility. If the truth of this hypothesis be confirmed, light will probably be shed not only upon the reactions of muscles under various circumstances, but also upon their behaviour in many diseases. For instance, it has

been suggested that the reaction of degeneration is the result of disappearance of fibrillar substance, accompanied by an undue increase of sarcoplasmic substance, so that the reaction is that of sarco-plasm; and the hypothesis has also been brought forward that the weakness of muscles in certain "functional" disorders may be due to a lack of proportionate contraction of the two substances.

It seems likely that in the future the subject may have an important bearing on clinical medicine.

CHAPTER IV

ELECTRICAL REACTIONS

NORMALLY, contractions of a muscle can be obtained by stimulating its nerve or the muscle fibres themselves by the faradic (interrupted) or galvanic (constant) current.

If, with the interrupted current, one pole is put on some indifferent part of the body, and the other on the muscle to be tested, sharp contractions occur in response to the stimuli, and, if the latter succeed one another with sufficient rapidity, the muscle is tetanised. The maximum effect is obtained when the electrode is placed upon certain spots known as motor points.* It is a matter of indifference which pole is used.

Contractions from the galvanic (constant) current can also be obtained through the nerves or muscle fibres, but only when the contact is made or broken.

In this form of stimulation there is choice of pole to be observed, for contraction is most easily obtained on making contact between the muscle and the negative (kathode) pole, as expressed by the statement that KCC is normally greater than ACC.

When the communications between the muscle and its corresponding motor cells in the cord are broken, as they are in many lower neuron lesions, certain distinctive modifications known as the reaction of degeneration are apt to occur.

D

33

*KCC > ACC in health.
ACC > KCC in disease*

The main features of this change are:

- (1) AS TO THE NERVE.—Gradual loss of excitability to both faradic and galvanic current, so that stimulation ceases to produce any result.
- (2) AS TO THE MUSCLE.—To faradic stimulation, at first a slow contraction, and later none at all. To galvanism the muscle may be, for a time, unduly irritable, but contracts slowly and in an abnormal manner to the two poles. The contraction is now more easily obtained by establishing contact with the positive (anode) pole, and thus ACC gives a greater effect than KCC. If no recovery occurs the excessive irritability vanishes, and in the end no reaction to any form of stimulation can be obtained.

When the fibres of a muscle do not waste uniformly, the reaction at any given time will be proportionate to the number of healthy fibres remaining, and thus there will probably be a diminished reaction to both currents, with no such qualitative changes as constitute the reaction of degeneration.*

Therapeutic uses of electricity.—In diseases of the nervous system electricity is chiefly used (1) as a general tonic, (2) to keep up the vitality of wasting muscles, (3) to relieve pain.

As a general tonic it is used as an adjunct to other forms of treatment in hysteria, neurasthenia, and nervous debility of any kind, and the choice lies between the interrupted, constant, and high-frequency currents.

The interrupted and the high-frequency currents are best when general stimulation is required. They appear to produce a certain amount

* Other less common variations in the electrical reactions are alluded to under the headings of the diseases in which they occur.

of improvement in nutrition, partly, no doubt, by increasing the activity of the vasomotor apparatus. They also act locally as a counter-irritant.

For the relief of pain the constant current is mostly employed. The positive pole (anode) should be applied to the painful part, since it has been shown to produce a greater sedative effect than the negative pole (kathode). When the two poles are kept stationary (the so-called *stabile* application) the current is less stimulating than when the electrode is moved (the so-called *labile* application); this fact therefore enables a further modification to be made in treating painful states. The pains of neuritis, sciatica, and various other forms of peripheral nerve lesions are often relieved by electrical treatment, and neuralgias and headaches are occasionally dispersed by the application of high-frequency currents.

In treating disorders of sensation, *e.g.*, the anæsthesias of hysteria, the application may often best be made by a wire brush.

The maintenance of the nutrition of wasting muscles is one of the most valuable functions of electricity. For this purpose the interrupted current is generally the most useful. An idea has been prevalent that the interrupted current should be applied only when the muscle contracts to it, and that in other cases preference should be given to the constant form, but Dr. Lewis Jones has shown that there is no adequate reason for holding strictly to this view. Moreover, cases showing the reaction of degeneration seem to have progressed quite as well with the one as with the other.

This question of the choice of current is important, since it is usually far simpler for the patient to use and maintain a faradic battery.

The application of static electricity is credited with producing improvement in metabolism, but in this country little therapeutic use is made of this form.

CHAPTER V

LUMBAR PUNCTURE

Technique.—In making a lumbar puncture the interval between the third and fourth or between the fourth and fifth lumbar vertebræ should be selected. A line joining the highest points of the iliac crests passes through the fourth lumbar spine, so that the puncture should be made either above or below the line, midway between the spinous processes, and half an inch or rather less from the middle line. It is scarcely necessary to say that the skin, the needle, and the hands of the operator should be thoroughly sterilised. The skin over the chosen spot should be frozen, but in the case of children a general anæsthetic is sometimes necessary.

The needle, which in the case of an adult should be about three and a half or four inches long, and in the case of an infant about two and a half inches, should have the calibre of an ordinary needle for the injection of antitoxin.

Position.—The patient should lie on the side, with the knees drawn up and the back arched outwards. When all is ready, the needle is inserted forwards and slightly inwards, and if it strikes upon bone it must be withdrawn a little and its direction somewhat changed. There is no great difficulty in discriminating between bone and ligamentum subflavum, which also offers some resistance.

If the pressure is normal the fluid flows out

drop by drop when the point of the needle is in the arachnoid space, but if the pressure is increased the flow is more forcible. Occasionally a little blood from some small vessel is mixed with the first few drops, but this soon disappears. Some operators prefer to use a syringe as a handle to the needle, but this is not necessary, and only in exceptional cases should any suction power be used to withdraw the fluid.

Value in diagnosis.—For ordinary purposes of diagnosis about a drachm of the fluid is sufficient, and it should be received into a sterilised test tube. This may be chemically examined, and,

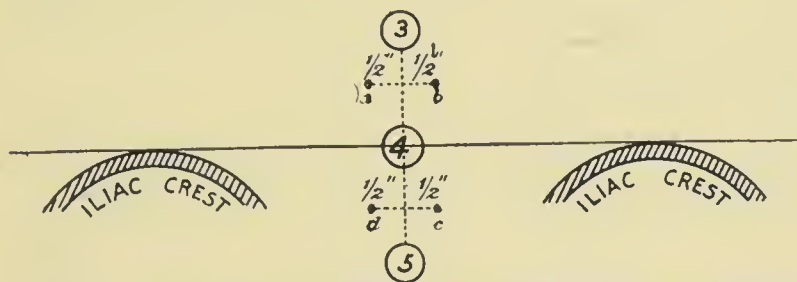


Fig. 6.—Diagram showing positions suitable for lumbar puncture.
(H. MacCormac.)

after it has been centrifugalised, slides for the microscope* and cultivations of micro-organisms can be made.

Normal cerebro-spinal fluid is quite clear, and when examined under the microscope contains no cellular elements, or at most a very few lymphocytes.

In meningitis the fluid is often turbid, though in the tuberculous variety it may remain clear. In addition to bacteriological evidence of disease, microscopic examination shows the presence of lymphocytosis, and in the non-tuberculous varieties, such as epidemic cerebro-spinal, streptococcal, and pneumococcal meningitis, polymorphonuclear cells also are generally present.

* For these Jenner's stain or methylene blue may be used.

The reaction for sugar, which can be obtained from normal fluid, may be diminished or absent in meningitis.

Lymphocytosis can often be demonstrated in the early stages of general paralysis of the insane, tabes, and syphilitic diseases of the nervous-system. It has also been found in disseminated sclerosis and anterior poliomyelitis. Undue stress must not be laid on this method of diagnosis, and in all doubtful cases the results should be carefully considered in conjunction with the other signs before a decision is made.

Value in therapeutics.—The therapeutic value of lumbar puncture lies in the relief of intracranial pressure, though in most cases its effects are, unfortunately, only transient. It has, for instance, been found useful in cases of cerebral tumour and meningitis, and also in cases of coma from renal disease and injuries to the skull.

Some unpleasant symptoms, such as headache, nausea, and giddiness, occasionally follow lumbar puncture, and it is always advisable for the patient to rest for some hours after the operation. Care must also be taken not to draw off too much fluid; the amount naturally varies with the pressure of the fluid and the conditions for which it is drawn off, but if this point be borne in mind it is not likely that an excessive amount will be withdrawn.

SECTION II.—THE PERIPHERAL NERVES

CHAPTER VI

PARALYSIS OF CRANIAL NERVES

I.—THE FIRST, OR OLFACTORY NERVE

THE olfactory apparatus comprises the following elements: (1) the olfactory cells, situated in the olfactory mucous membrane, the central processes of which are continuous with the fibrils of the olfactory nerves proper. The latter end in (2) the olfactory bulbs, by arborising round the termination of one of the dendrites of the so-called mitral cells, situated more deeply in the substance of the bulb. The centrally-directed mitral axon is then conducted along (3) the olfactory tracts, which are each connected by means of a mesial and a lateral root with (4) the uncus, the cortical centre for smell. The olfactory apparatus thus consists of a series of relays of neurons: the olfactory cells, the mitral cells of the bulb, and the cortical cells of the uncinata convolution.

The olfactory nerves proper, about twenty in number on each side, pass through the cribriform plate of the ethmoid, and tend to be arranged in an inner and an outer group; the fibrils of the former are distributed over the upper part of the ethmoidal septum, and those of the latter supply the mucous membrane overlying the superior turbinated bone. The peripheral process of the olfactory cells, of which these nerve fibrils are the central offshoots, ends on the surface of the olfactory mucous membrane, and is surrounded by a tuft of short filaments, the olfactory hairs.

1 olfactory cell

2 olf. bulbs.

3 olf. tracts

4 Uncus.

To test the sense of smell, aromatic substances, *e.g.*, cloves and peppermint, should be applied in turn to each nostril, the other being meanwhile closed. Substances which stimulate the fifth nerve, *e.g.*, ammonia, must not be used, otherwise the sensations thereby produced may be mistaken for those of smell. The local causes of perversion or loss of this sense are very numerous, and must be excluded before the trouble can be attributed to the olfactory nervous apparatus. It should be remembered, too, that olfactory sensations may form the aura of epileptic fits, and may also occur in association with tumours of the uncinate convolution which is the cortical centre for smell.

In hysteria also the sense of smell is frequently lost, usually on the same side as the hemi-anæsthesia, but occasionally the loss may be bilateral.

II.—THE SECOND, OR OPTIC NERVES, AND VISUAL PATHS

The optic nerve contains the visual fibres, and also serves as the afferent path of the pupil reflex to light.

Apart from disease of the structures of the eye itself, the sight may be impaired from lesions which interfere with the visual fibres at any part of their course. These may be grouped according as they are situated in—

1. The optic nerves.
2. The optic chiasma.
3. The fibres between the chiasma and cortex.
4. The cerebral cortex.

Anatomical.—The optic nerve runs from the eyeball in front to the optic commissure behind, where a partial decussation of its fibres with those of the opposite side takes place. The nerve, in its course, traverses the orbit, the optic foramen, and the anterior fossa of the skull, and is invested by

meningeal sheaths continuous with the dura and pia arachnoid.

The nerve is connected with the globe of the eye at a point one-eighth of an inch on the mesial side of the axis of the eyeball, and after piercing the tunics of the latter spreads out at the optic disc to form the innermost layer of the retina.

The chiasma, or commissure, situated at the base of the brain, is adherent to the floor of the third ventricle, and rests upon the olivary eminence of the sphenoid bone; it is situated just anterior to the tuber cinereum, whilst at the sides the internal carotid arteries ascend to the brain.

At the commissure a partial decussation of the fibres of the two optic nerves takes place; thus, the fibres originating in the mesial half of each retina cross at the chiasma to join the optic tract of the opposite side, but those arising in the temporal portion of the retina do not cross the mesial plane, but are continued into the tract of the same side. These fibres, which are situated in the posterior part of the chiasma, are, in all probability, not connected with vision, but constitute a commissural bundle running on each side into the mesial root of the optic tract, and

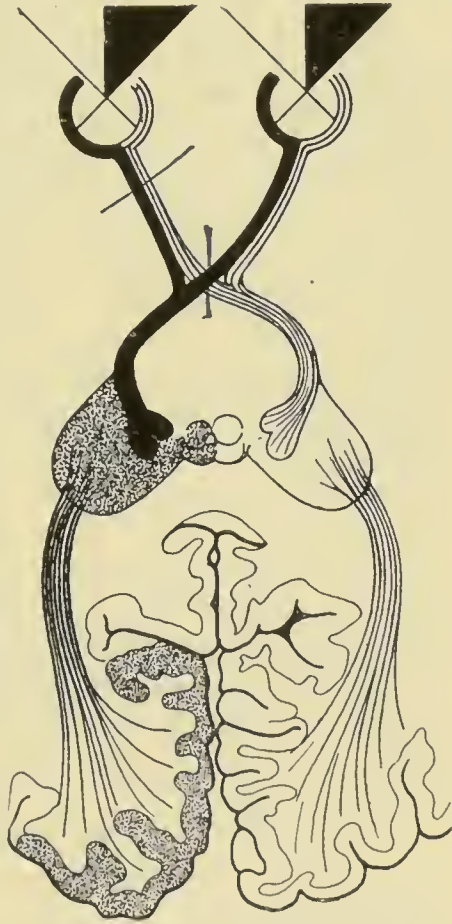


Fig. 7.—Diagram of the visual paths.
(After Déjérine.)

connecting the two internal geniculate bodies together. This is known as the commissure of Gudden.

The optic tracts proceed backwards around the crura cerebri, and each divides into a mesial and a lateral root. The mesial root has been referred to above, and is probably not connected with vision; the lateral root ends in the superior quadrigeminal body, the external geniculate body, and the pulvinar of the optic thalamus, which constitute the lower visual centres.

The cortical connections.—These lower visual centres are connected with the cerebral cortex by a large strand of fibres, known as the optic radiation. These fibres lie in the retroventricular part of the posterior limb of the internal capsule, and sweep backwards into the occipital lobe on the outer side of the posterior horn of the lateral ventricle.

Other connections of the lower visual centres are—

(a) With the nuclei of the ocular nerves—probably this connection is established through the posterior longitudinal bundle.

(b) The superior quadrigeminal body is connected by the mesial fillet with the medulla and cord.

1.—LESIONS OF THE OPTIC NERVES

OPTIC NEURITIS

Excluding local conditions in the eyeball itself, the causes of optic neuritis may be divided into two classes, according as they arise from (a) intracranial disease or (b) general toxæmias.

(a) In **intracranial disease**, meningitis, tumours, and abscesses are the commonest causes of optic neuritis. Some faint signs of inflammation may occasionally precede the more prominent condition of atrophy in disseminated sclerosis, tabes, and general paralysis of the insane. In

meningitis some degree of optic neuritis frequently accompanies the tuberculous and streptococcal varieties. It is more seldom met with in the cerebro-spinal and posterior basic varieties.

Inflammation of the optic discs is the most constant sign that accompanies cerebral tumours. It occurs in about 90 per cent. of the cases, and is of very great diagnostic importance.

With abscess of the brain, neuritis, though frequent, is not so constant as in tumours, nor when present is it usually so intense.

(b) **General toxæmias.**—The toxæmias that accompany chronic renal disease, intense anæmias, diabetes, chronic lead poisoning, and occasionally other infections, may all give rise to optic neuritis.

Ophthalmoscopic appearances.—In the early stages the disc is hyperæmic and its borders are ill defined. As the process increases the physiological cup becomes filled in and the course of the arteries at its edges becomes obscured by inflammatory exudate. At the same time the disc becomes swollen. Later on, changes appear in the retina. The veins become greatly dilated, and small flame-shaped hæmorrhages take place into the retinal tissue. Lastly, the exudate is partially absorbed and partially organised into connective tissue; the optic nerve becomes white, the vessels small, and failure of sight is then permanent.

Symptoms.—It is very important to note that there is often little or no impairment of vision in the early stages of optic neuritis, and that systematic ophthalmoscopic examination is the only way by which overlooking its presence can be avoided.

RETROBULBAR NEURITIS

In retrobulbar neuritis there is an inflammation followed by degeneration of the axial fibres (i.e., the macular bundle). It is this condition that gives rise to the toxic amblyopias from tobacco and occasionally other poisons.

Symptoms.—Vision is gradually impaired, especially for colours, so that the earliest signs are usually those of a central scotoma for green and red. For a time there may be no changes to be observed with the ophthalmoscope, but later there is some pallor of the disc, especially in its temporal half.

OPTIC ATROPHY

Optic atrophy may occur secondarily to the conditions enumerated under neuritis, or it may be due to a primary degeneration of the nerve fibres.

Primary atrophy is principally met with in tabes dorsalis, disseminated sclerosis, and general paralysis of the insane. The toxic amblyopias, as stated above, are probably examples of axial neuritis.

In the primary atrophy of tabes the disc gradually becomes paler, the vessels undergoing but little change. Vision gradually fails, and the contraction of the colour field is at first generally greatest.

HEREDITARY OPTIC ATROPHY (LEBER'S DISEASE)

The condition rarely met with and known as hereditary optic atrophy was first described by Leber. The disease, which is characterised by atrophy of the optic nerve, tends to run in families and is usually transmitted by the females and inherited by the males. It sometimes skips one generation, to appear again in the next. In the majority of cases the symptoms first appear in young adults (age from 15 to 20), but they have been observed to begin both in children and elderly people.

The patients complain of failure of sight, and the ophthalmoscopic appearances are those of partial atrophy, preceded, possibly, by some slight signs of neuritis. The morbid changes occur in both eyes, but in most of the cases they do not

progress far enough to cause complete blindness. The symptoms closely resemble those caused by tobacco, but in Leber's disease there is a central scotoma for white, whereas in tobacco amblyopia the failure of central vision is chiefly for red and green.

It seems probable that the pathological changes closely resemble that of tobacco amblyopia, and that the condition is in reality one of axial neuritis (Nettleship).

There is no adequate form of treatment known.

2.—LESIONS OF THE CHIASMA

The chiasma is liable to be damaged by tumours at the base of the brain, especially by those connected with the pituitary body; hence it often happens that patients with signs

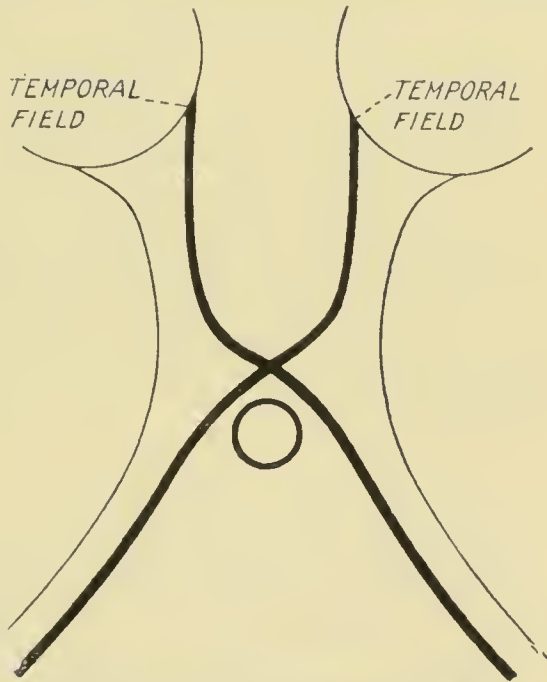


Fig. 8.—Diagram showing probable effects of pressure on the chiasma.

of acromegaly complain first of their vision. Reference to the diagram (Fig. 8) shows that the effects most likely to be produced by pressure on the chiasma are due at first to impairment of the function of those fibres which go to the inner side of each retina and supply the temporal field of vision, and later to the extension of pressure on to the whole trunk of one or both nerves. The symptoms will, in such a case, begin with double temporal hemianopia, and may end in complete atrophy.

It is possible, though naturally rare, in the

process of atrophy of the visual fibres at the commissure, or owing to the symmetrical position of two tumours, that the fibres proceeding to the outer half of each retina may be caught, while those going to the inner halves are left intact. In that case, the function of the outer half of each retina being lost, there will be blindness in both nasal fields, *i.e.*, a double nasal hemianopia.

3.—LESIONS BETWEEN THE CHIASMA AND CORTEX

If the fibres are implicated anywhere in their course between the commissure and the cortex, *e.g.*, in the neighbourhood of the internal capsule, then the outer part of one half of the retina and the inner part of the other will be affected—*i.e.*, the temporal field on one side, and the nasal field on the other,—and thus a simple or homonymous hemianopia, which is by far the commonest variety, is produced.

4.—LESIONS OF THE VISUAL CEREBRAL CORTEX

If the cuneate portion of the occipital lobe is diseased the result will again generally be homonymous hemianopia, since the fibres start from this part of the cortex to supply one half of each eye. Occasionally, however, a quadrantic hemianopia occurs. Lastly, if the area of the cortex known as the supramarginal and angular convolutions is diseased, the result may be that known as "crossed amblyopia," *i.e.*, a concentric diminution of both fields of vision, but more marked on the side opposite the lesion. Such a condition may be associated with visual aphasia, *i.e.*, inability to recognise the significance of written words, sometimes also spoken of as "word-blindness." Crossed amblyopia is rare in organic disease, but is the disorder of vision most commonly met with in hysteria.

Wernicke's hemiopic pupil reaction.—This test

is carried out by throwing a narrow beam of bright light on to the blind half of a hemiopic retina and noting if the pupil contracts.

Absence of any contraction is an indication that the lesion causing the hemianopia is on the peripheral side of the pupil centre, *i.e.*, somewhere between the optic tract and the corpora quadrigemina, while the presence of contraction shows that the lesion is behind the centre for the pupil, *i.e.*, in the optic radiation or occipital cortex.

The test is of some value as an aid in localising cerebral tumours, in which hemianopia is one of the symptoms, but it is not an easy one to perform.

III.—OCULAR NERVES

The *third, fourth, and sixth* nerves may conveniently be considered together, since they all supply the muscles of the eye.

THE THIRD NERVE

Anatomical.—The nucleus of this nerve is situated in the floor of the aqueduct of Sylvius. The fibres emerge on the surface of the inner side of the crus cerebri at the superior border of the pons, and, passing into the cavernous sinus, finally enter the orbit through the sphenoidal fissure and supply all the muscles of the eyeball except the superior oblique (4th) and the external rectus (6th). This nerve also innervates the levator palpebræ, the ciliary muscle, and the sphincter of the iris. The nerve enters the orbit in two divisions: the superior supplies the levator palpebræ and superior rectus, the inferior is distributed to the other muscles.

The dilator of the iris gets its nerve supply from the sympathetic.

Lesions of the third nerve.—The whole of the third nerve or any of its branches may be affected. When the nerve is completely paralysed the upper lid drops (ptosis) from the loss of power

3rd.
Lev. palpe.
ciliary.
Sphincter Iris
4th.
Sup. oblique
6.
Ext. Rectus
Sympathetic
Dilator Iris

of the levator palpebræ, and the endeavour that is being made to keep the eye open is often reflected in the furrowed forehead which arises from the contraction of the occipito-frontalis.

The eyeball is pulled outwards by the un-antagonised external rectus, and the power of moving it inwards, upwards, and downwards is also lost; all that can be managed is a slight movement downwards and inwards, produced by the superior oblique. The pupil is dilated and unable to react to light or during accommodation.

When, as often happens, a part only of the nerve is paralysed, one or more of the muscles which it supplies suffer.

In paralysis of the superior rectus the upward and internal rotatory movements of the eyeball are impaired. The resulting diplopia is crossed, and the false image is above and tilted away from the true one. When the inferior rectus is paralysed the movements of depression and external rotation are impaired. The diplopia is crossed, and the false image is tilted towards the true one.

In paralysis of the inferior oblique the upward and external rotatory movements are weakened. The diplopia is homonymous, the false image being above and tilted away from the true one.

Congenital ptosis is sometimes met with, and is probably due to maldevelopment of the levator palpebræ. It is often associated with a deficient power of moving the eye upwards.

Another class of cases, also of congenital origin, is that known as "jaw-winkers." In these there appears to be a deficiency in the nervous innervation of the levator palpebræ, while at the same time there is an association of movement between it and the muscles of mastication. The result is that of ptosis while the jaw muscles are at rest, and an opening and closing of the eyes when masticatory movements are going on.

THE FOURTH, OR TROCHLEAR NERVE

Anatomical.—The nucleus of the fourth nerve is situated in the floor of the aqueduct of Sylvius, just behind that of the third, and the fibres arising from it decussate in the superior medullary velum with their fellows of the opposite side. On emerging at the surface it winds round the crus cerebri, lying between the posterior cerebral and superior cerebellar arteries, and, after passing through the cavernous sinus, enters the orbit through the sphenoidal fissure to supply the superior oblique, which moves the eyeball downwards and outwards with a wheel-like rotatory movement inwards.

Lesions of the fourth nerve.—Defective movement from weakness of this nerve is not usually obvious. The chief symptom is diplopia on attempting to look downwards, as in coming downstairs. Giddiness frequently accompanies paralysis of the fourth nerve, owing to the type of the diplopia.

—The diplopia is homonymous, and the false image is on a lower level and obliquely inclined towards the true image.

THE SIXTH NERVE, ABDUCENS

Anatomical.—The nucleus is in the floor of the fourth ventricle, on a level with the lower part of the pons.

The fibres emerge from the groove between the pons and the medulla. Passing through the cavernous sinus, the nerve enters the orbit through the sphenoidal fissure, between the two heads of the external rectus, and terminates in this muscle.

In paralysis of this nerve there is deficient movement of the eyeball outwards, and when the loss of power is complete the eyeball cannot be moved beyond the middle line.

There is internal strabismus owing to over-action of the internal rectus, which in long-standing cases may become permanently contracted.

There is diplopia on attempting to look outwards on the paralysed side. The diplopia is homonymous, and the true and false images are parallel.

If there is a lesion of the nucleus of the sixth nerve there will be paralysis of the internal rectus of the opposite eye, since its movements are governed through fibres from the nucleus of the sixth:

Nystagmus.—Allusion may be briefly made here to those oscillations and jerky movements known as nystagmus, which will again be referred to under the headings of the different diseases in which it occurs.

Nystagmus occurs in various organic diseases, *e.g.*, in disseminated sclerosis, Friedreich's disease, and in lesions of the cerebellum. It is also met with as a temporary affection in children who are the subjects of "nodding spasms," in cases of paresis of the ocular muscles, and also in miners, who are supposed to develop it in consequence of the continuous strain to which their eyes are put. The movements of the eyeballs may be from side to side (lateral nystagmus), in which case they are usually best brought out by fixing the eyes to the right or left; or they may be rotatory, and visible in all positions of the eyeball.

In cases where the diagnosis is doubtful, the presence of nystagmus is frequently an important confirmation of the presence of organic disease, and more especially is this so in the early stages of disseminated sclerosis.

The general effects of **paralysis of the ocular muscles** may be summed up as follows:

1. **Limitation of movement.**—When a muscle is paralysed the movement of the eyeball is limited in the direction in which the muscle acts. This can be shown by asking the patient to follow an object with his eyes, when the results of loss of power in one or more directions will usually be manifest.

2. **Squint.**—When one or more muscles are

paralysed, not only is there limitation of movement in certain directions, but the balance of power when the eyeballs are at rest is also lost, and the contraction of the unopposed muscles causes the eyeballs to deviate so that their axes cease to be parallel, thereby producing the condition known as *strabismus*, or *squint*.

3. **Diplopia.**—Double vision is the result of the strabismus. The eyeballs having ceased to be parallel, the images no longer fall on corresponding parts of the retina, and so two objects appear to be present. It is shown by making the patient follow an object in different directions, and then, as soon as the affected eye ceases to fix that object, two images will be seen. Double vision does not, however, necessarily accompany every case of squint, as the patient sometimes learns to disregard the false image.

When the false image is on the same side as the affected eye the condition is known as simple or *homonymous diplopia*, but when the images cross so that the one formed on the retina of the affected eye is projected in such a manner that it is opposite the sound eye the condition is known as *crossed diplopia*.

In all cases of internal squint—convergent strabismus—the diplopia is simple. If, for instance, the external rectus of the left eye is paralysed, the image will fall upon the retina to the inside of the yellow spot, and in the field of vision it will appear to the left of the image belonging to the right eye.

In all cases of external squint—divergent strabismus—the diplopia is crossed. Thus, when the axes of the eyes are prolonged forwards and the lines intersect the diplopia is simple, and when they diverge the diplopia is crossed.

In cases of paralysis of the internal and external recti the true and the false images will be parallel to one another, but in paralysis of the

other muscles rotation of the eyeball comes into play and the false images become tilted.

4. **Secondary deviation** is an over-action of the sound eye, and is demonstrated as follows: If the sound eye is covered up, and the patient made to "fix" an object with the paralysed eye in the direction of the weak muscle, the eyeball turns as far as it can in the desired direction, and at the same time the sound eye moves over a still greater distance in the same direction.

If a man have a weak external rectus on the left side, and his sound eye be covered up, and he be made to fix an object towards his left-hand side with the left eye, the latter will then attempt to turn as far as it can with its weak external rectus, and if the sound eye be watched it also will be seen to turn in the same direction, but the balance between them is upset, so that instead of the two remaining parallel, the sound eye rotates through a larger arc than the weak one. This is due to the fact that the left external rectus and the right internal rectus always act together: both receive the same amount of stimulus to look to the left, but on account of the weakness of the external rectus on the left side the stimulus, in order to effect the movement, must be stronger than is usually necessary, and the internal rectus of the right eye, being sound, responds to this extra stimulus, and so overacts and moves the eyeball over a distance which is out of proportion to that of the left.

5. **Erroneous projection of the field of vision.**—This symptom depends on the limitation of movement of the eyeball, so that objects appear in one spot when they are in reality in another. It is due to the fact that the relations between objects and ourselves are judged by the range of movement of our eyes, and when this range of movement is suddenly upset the strong attempt which is made to move the eye in the direction

of the weakened muscles occasions the idea that the eyeball has moved farther than it really has, and thus upsets calculations of distance which have been based on all previous experience.

If the external rectus on the left side be weak, then on turning the eye to the left there is an increased innervation of the weak muscle in order to try to produce the movement. As the individual has been in the habit of judging the amount of movement by the amount of innervation necessary to cause it, he naturally interprets this increased innervation as producing its full effect, and therefore he thinks his eye has turned farther to the left than it really has, and, as a consequence, objects appear to him to be farther to the left of himself than they really are.

IV.—THE FIFTH, OR TRIGEMINAL NERVE

Anatomical.—This nerve is partly motor and partly sensory in function. The **motor fibres** arise from the motor nucleus which lies in the grey matter at the lateral angle of the fourth ventricle, and from the descending or mesencephalic root of the trigeminal, situated in the grey matter surrounding the Sylvian aqueduct. The small motor root passes forwards beneath the Gasserian ganglion, and is wholly incorporated in the inferior maxillary division of the trigeminal. It innervates the temporal, masseter, the external and internal pterygoid, the mylohyoid and anterior belly of the digastric, possibly also the levator palati and azygos uvulæ (through Meckel's ganglion), and the tensor tympani and tensor palati muscles (through the otic ganglion).

Lesions of the motor root.—Lesions of this root are followed by loss of masticating power, and the lower jaw, when thrust forward, is tilted to the weakened side by the unantagonised healthy muscles.

Temporal
masseter
Ext. Pterygo
Int. Pterygo
mylohyoid
ant. b. Digastric
Levator palati
Azygos uvulæ
Tensor Tympani
Tensor Palati

The **sensory root of the fifth nerve** ends in two nuclei of termination: (a) the sensory nucleus of the trigeminal, situated in the pons, and (b) the spinal root, connected with the substantia gelatinosa of Rolando.

The sensory root passes with its motor companion across the base of the brain and enters the Gasserian ganglion, which rests on the apex of the petrous portion of the temporal bone.

Thence the fibres separate into three great divisions (ophthalmic, superior and inferior maxillary), which supply sensation to the anterior portion of the skull, half of the face, including the orbit and eyeball, nose and nasal cavity, lips, teeth, mouth, and the anterior two-thirds of the tongue.

Neuralgia is very common in the area of distribution of one or more branches of this nerve, and division of the branches of the latter gives rise notably to anæsthesia, but often also to disturbances of nutrition of the skin, and especially of the eye—ulceration of the cornea and conjunctiva, and suppuration of the whole eyeball, all being possibilities. The most prominent trophic disturbances that occur over the area supplied by this nerve are, perhaps, those connected with facial hemiatrophy.

In spite of all these disorders of nutrition attending injury of the nerve, it does not necessarily follow that special trophic fibres exist. Many of the effects, especially those connected with the eye, can be accounted for by the loss of that protection against harmful stimuli which integrity of the sensory nervous system confers.

The fifth nerve makes many important connections, and the peripheral course of the fibres subserving the sense of taste is a somewhat complicated one. Two main descriptions of the course of these taste fibres are current:

1. It is held that the taste fibres from the

anterior two-thirds of the tongue run at first in the lingual nerve, then in the chorda tympani, to the geniculate ganglion of the facial; subsequently by the great superficial petrosal nerve to Meckel's ganglion and the superior maxillary division of the fifth. The taste fibres from the posterior third of the tongue run at first in the glosso-pharyngeal as far as the petrous ganglion, then, by way of Jacobson's nerve, to the tympanic plexus, from which they follow the course of the small superficial petrosal nerve to the otic ganglion, and thus to the inframaxillary division of the trigeminal.

2. Edinger regards the nucleus of taste as a mass of grey matter, co-extensive with the sensory nucleus of the fifth, which receives certain afferent fibres: (a) from the lingual nerve, through the medium of the Gasserian ganglion; (b) from the chorda tympani, through the geniculate ganglion of the facial, and the pars intermedia; and (c) from the glosso-pharyngeal, through the petrous ganglion (Box and Eccles).

FACIAL HEMIATROPHY

Facial hemiatrophy (Fig. 9) is a rare condition, and appears to depend upon a disturbance of nutrition in the area of distribution of the trigeminal nerve. It generally first shows itself during childhood, and is more commonly found in females than in males.

Symptoms.—The earliest signs are often found in the skin, which becomes shiny and pigmented with yellowish and brownish patches. The connective tissue soon takes part in the atrophy, and finally the growth of the whole side of the face, including the bones, is retarded. The muscles do not undergo any active atrophy, but they naturally waste from disuse and their movements are impeded by the condition of the tissues around.

The hairs of the eyebrows often turn grey or drop out, and the hair of the head on the diseased side is sometimes also affected. The cartilage of the ear is usually wasted, and the corresponding half of the tongue is sometimes atrophied, as seen



Fig. 9.—Hemiatrophy of the left side of the face and of the left half of the tongue.

in the illustration (Fig. 9); and here again it is the fat and connective tissue, not the muscles, that disappear. There may be deficient sweating on the affected side; the teeth are often brittle.

There is usually no tendency for the atrophy to spread, though occasionally it extends to the structures on the other side of the face. There

is very seldom any loss of sensation, but neuralgic pains may be felt over the wasted area.

Pathology.—The area to which the atrophy is generally confined has naturally led to a suspicion that some affection of the fifth nerve is at the root of the trouble. Sclerotic changes have been found in the nerve trunk, but it is difficult on this assumption to understand how the sensory fibres escape so completely.

The possibility of the atrophy being due to changes in the sympathetic has been suggested, but there is very little evidence to support it, except that dilatation of the pupil on the affected side has been noted, and this was well marked in the case illustrated in Fig. 9.

The most plausible idea is that which regards the condition as a tropho-neurosis, due to a failure of the trophic function of the fifth nerve to the exclusion of its sensory and motor functions. This explanation, however, assumes the existence of special trophic fibres, of which there is no adequate proof.

Treatment.—The wasted parts cannot be restored. All that can be done is to maintain the nutrition, general and local, at as high a level as possible, and thereby to try to prevent the disease from extending.

V.—THE SEVENTH, OR FACIAL NERVE

Anatomical.—This nerve arises from a nucleus in the pons below the floor of the fourth ventricle. The fibres are at first in close relation with the nucleus of the sixth nerve, around which they wind. This fact is of value in the localisation of cerebral lesions, since their proximity makes the two nerves very liable to suffer together from any gross disease in their neighbourhood.

The facial fibres appear at the surface of the brain at the angle between the pons and cerebellum, and in company with the auditory nerve,

but, separated from it by the pars intermedia of Wrisberg, they pass across the base of the brain and enter the internal auditory meatus. Here the facial leaves the auditory nerve and enters the aqueductus Fallopii, through which it passes, and finally leaves the skull by the stylo-mastoid foramen to supply the muscles of half the face and the platysma.

During its passage through the aqueductus Fallopii the nerve swells out into the geniculate ganglion, from which spring the greater, lesser, and external superficial petrosal nerves, and it also gives off, while passing through the canal, a branch to the stapedius and the chorda tympani, which last, leaving the main trunk a little above the stylo-mastoid foramen, turns forwards, and, after passing through the tympanum and communicating with the otic ganglion, joins the lingual nerve, and so is conducted to the tongue to supply the anterior two-thirds with sensation of taste, the posterior third being supplied by fibres from the glosso-pharyngeal. (*See above.*)

PERIPHERAL FACIAL PARALYSIS (BELL'S PALSY) *

Etiology.—The principal causes of peripheral facial palsy may be grouped according to the situation of the lesion.

- (1) *In the pons.*—The facial nucleus may be injured by tumours, or, more rarely, it may be the seat of an acute inflammatory process and form part of an acute anterior poliomyelitis. Nuclear degenerations are sometimes met with in the course of cases of progressive bulbar paralysis, tabes, and disseminated sclerosis.

* Strictly speaking, "Bell's palsy" includes only that form in which the lesion occurs after the nerve has left the stylo-mastoid foramen, but the term is frequently used for all forms of peripheral lesions.

- (2) At the base of the brain.—As the facial nerve passes over the base of the brain to reach the internal auditory meatus it may again be compressed by tumours, especially by those which grow in the angle between the pons and the cerebellum. It is also apt to be injured by gummatous and other forms of meningitis, and by fractures of the base of the skull.
- (3) In the bony canal.—When in the canal the nerve may be injured from disease of the bone, which so frequently arises in connection with suppuration of the middle ear. It is also whilst within the unyielding walls of the canal that the nerve is so liable to swell and be compressed, as the result of cold and toxæmias.
- (4) Outside the canal.—After the nerve has emerged from the stylo-mastoid foramen it may be injured by tumours of the parotid region, or by wounds of the face.

In the majority of the cases the lesion is situated within the canal, and is thought to be of a “rheumatic” nature. The history most often given is that the paralysis came on after being exposed to cold, or more especially a draught such as may occur from the windows of a railway train. It is generally considered that inflammation of the nerve sheath is the primary lesion, and that the consequent swelling, having no room to expand in the bony canal, compresses the nerve fibres. If this hypothesis is correct these “rheumatic” cases may be regarded as being analogous to many cases of sciatica where an inflammation of the nerve sheath is also frequently the primary lesion, but it is, of course, possible that different toxins give

rise to the paralysis in different cases, for the occasional extension of the paralysis to the facial nerve in a general neuritis (*e.g.*, from alcohol or diphtheria), though rare, shows that the facial nerve is not immune from the poisons which are



Fig. 10.—Right-sided facial paralysis of peripheral type. Note the inability to close the eye and the obliteration of the nasolabial fold on the paralysed side.

more apt to injure the nerves of other parts of the body.

Symptoms.—Peripheral lesions (*i.e.*, lesions of the lower neuron situated at or below the nucleus) of the trunk of the facial nerve result in paralysis of the muscles supplied by that nerve, with some additional symptoms according to the locality of the lesion.

The paralysis being of the lower neuron type, the loss of power is accompanied by wasting and changes in the electrical reactions. The paralysed side of the face looks somewhat flattened (Fig. 10); the nasal fold is obliterated, and the angle of the mouth drops, giving to the patient the idea that it is "drawn up" on the sound side; the nostril on the affected side is somewhat smaller.

1. Loss of power.—In a well-marked case there is loss of power of all the muscles on one side of the face, and this may be demonstrated by asking the patient to perform the following actions:

(a) To look up to the ceiling. The forehead will remain smooth owing to paralysis of the occipito-frontalis.

(b) To frown. There will be no furrow, owing to loss of power in the corrugator supercilii.

(c) To close the eyes. The eye on the affected side cannot be closed, and the attempt to close it is accompanied by a rolling upwards and inwards of the eyeball.

This failure to close the eye is due to weakness of the orbicularis palpebrarum, and owing to the laxity of the fibres of this muscle in the lower lid the tears are apt to run down the face (epiphora). Conjunctivitis from irritation of the unclosed eye may also arise.

(d) To show the teeth. The mouth does not move towards the paralysed side.

Other symptoms that may be noted are:

Weakness of the buccinator muscles, causing a tendency for the food to stagnate between the cheek and the gums.

Affections of hearing. In some cases where the nerve to the stapedius has been paralysed, hearing has been noted to be more acute than usual, due, it is thought, to the unantagonised action of the tensor tympani tightening the drum.

2. Loss of taste.—If the chorda tympani is affected, taste will be lost over the anterior two-thirds of the tongue on the same side as the facial paralysis.

It is tested by placing a little bitter or sweet substance (*e.g.*, quinine sulphate or sugar) on the



Fig. 11.—Bilateral facial paralysis. Note the inability to close the eyes and the general expressionless appearance of the face.

side of the protruded tongue, and asking the patient to hold up his hand if he tastes anything.

If he is allowed to speak or to move his tongue about, the substance is liable to be diffused and tasted on the opposite side and at the back of the tongue.

There is no loss of cutaneous sensation, but pain is sometimes experienced in the early stage of the paralysis.

Diagnosis.—There is not usually any difficulty in determining whether facial palsy is present. The only condition which may perhaps resemble it superficially is that of facial hemiatrophy, but in the latter the muscles, though rigid, are still capable of voluntary contraction, and it is easy to see that changes in the skin and connective tissue are the main sources of the trouble.

Having decided that paralysis is present, it is necessary to consider whether the cause lies in the muscles, the lower neuron of the nerve, or the upper neuron.

The muscles of the face undergo primary degeneration in some of the myopathies, and weakness is frequently met with in cases of myasthenia gravis, but neither of these conditions need be confused with peripheral facial paralysis.

In the myopathies the muscles chiefly affected are those at the angle of the mouth; both sides are affected, and in addition there will nearly always be some signs of wasting of the trunk muscles.

In myasthenia gravis it is unlikely that only one half of the face would be weakened, and, further, the variation of the signs, together with the ptosis and bulbar symptoms, make it very unlikely that serious difficulty would arise in that direction.

A primary origin of the muscular weakness being thus excluded, the next point to be decided is whether the lesion is in the lower or the upper neuron, and this it is usually possible to do by noting the distribution of the weakness; for whereas in lesions of the lower neuron there is generally paralysis of the whole of one half of the face, in those of the upper neuron the movements which habitually take place together on the two sides (bilateral movements) escape or are only weakened. Thus, whereas in lower neuron lesions the patient cannot close the eye and wrinkle the

brow, in upper neuron lesions both actions can usually be performed. If, however, there is still any doubt as to which neuron is affected, it may usually be dispelled by an examination of the electrical reactions.

Having settled that the lesion is situated in the lower neuron, one must next try to determine its exact position.

1. If the facial paralysis is accompanied by paralysis of the external rectus on the same side, it suggests (provided there is no reason for thinking that there is more than one focus of disease present) that the lesion is at the facial nucleus itself, for it is there that the sixth nerve comes into close proximity to the seventh.

2. If deafness is present, it may be that both auditory and facial nerves are injured while they are situated close to each other at the base of the brain; but care must be taken before this view is accepted, for it may be that the deafness is due to middle-ear disease, and that the facial paralysis depends upon necrosis of the surrounding bone.

3. If taste is lost, it is evident that the disease is situated in that portion of the nerve wherein the chorda tympani runs, *i.e.*, between the geniculate ganglion and a point a little above the stylo-mastoid foramen.

Prognosis.—The outlook in a case of peripheral facial paralysis must, of course, largely depend upon the cause. Those cases in which the neuritis is apparently of a “rheumatic” nature, and which appear to be caused by exposure to cold, for the most part do well.

In those which follow disease of the bone the result is more doubtful; while the prognosis in those cases produced by new growth or by complete severance of the nerve must depend upon the possibility of removing the tumour or of suturing the nerve-track.

In the so-called “rheumatic” cases which so

commonly come before us, an incomplete palsy is a favourable sign by showing that the intensity of the inflammation is not great, and such cases generally recover rapidly. When the palsy is complete the electrical reactions are a useful guide. If, in a week or ten days after the onset of the paralysis, reaction to the faradic current still persists, a steady return of power may be predicted with confidence; if, on the other hand, the paralysed muscles give a reaction of degeneration, recovery will be much slower and more uncertain; and, lastly, if all reaction to galvanism ceases, the outlook is bad.

Late results of facial paralysis.—If complete recovery does not take place the paralysed muscles often gradually undergo some contraction, which has the effect of making the sound side appear at first to be paralysed, since the angle of the mouth on the contracted side is drawn up. This contraction is probably accounted for by changes in the muscle fibres, and about the time that it is beginning to take place a curious irritability is sometimes observed, whereby the muscles, though paralysed to voluntary movement, will act in association with other muscles, thus showing that apparently some conductive power still remains, slight though it may be. This late contraction does not appear to occur in those cases where the continuity of the nerve has been completely severed.

Treatment.—The best method of treating the “rheumatic” cases is by regular faradisation of the muscles; their nutrition is thus maintained so that they are ready to react to the impulses as soon as the latter are able to pass along the nerve; possibly the nutrition of the nerve-endings is improved at the same time. When there is no reaction to the faradic current it is customary to apply galvanism, but it is doubtful whether the latter gives any better results, even

though the muscles react to it when they do not to faradism.

Internally, potassium iodide in doses of about five grains seems to be useful; and at the beginning, counter-irritation, in the shape of a blister behind the ear, sometimes appears to be of value.

Where the paralysis is due to necrosis of bone the treatment must still be conducted on the same lines, and in many cases the ultimate results are good, since the nerve is injured by extension of inflammation rather than by pressure of bone, and when the inflammatory process subsides power returns, provided that the nutrition of the muscles has been carefully maintained.

If there is no sign whatever of any return of power after, say, six months, the question of operative treatment may be considered, for it is sometimes possible to restore a certain amount of movement by severing the facial trunk and grafting the distal end of the peripheral portion on to the spinal accessory, or, better, on to the hypoglossal nerve. By this means some of the motor impulses passing down the hypoglossal to the tongue are transferred to the facial muscles, and a certain degree of improvement is thus effected. This method, however, is subject to the drawback that the face and tongue must move at the same time, though after a period these movements may tend to become dissociated.

Careful consideration is, however, needed before deciding to adopt this line of action. In many cases the deformity and inconvenience produced by the facial paralysis are both slight, and are preferable to the problematical benefits to be obtained from an operation which has its own very distinct risks.

Facial Spasm. Unilateral facial spasm may be met with. It is usually of reflex origin, and is then typically seen in cases of tic douloureux, but it may also be caused by irritation of the facial

nerve trunk. These spasms of local origin must not be confused with the tics which are described in Chapter XLII.

VI.—THE EIGHTH, OR AUDITORY NERVE

Anatomical.—The auditory nerve consists of two elements—1, the cochlear nerve, and 2, the vestibular nerve.

1. The cochlear nerve, on which is situated the ganglion spirale, divides directly into branches to (1) the macula aconstica of the saccule, (2) the ampulla of the posterior semicircular canal, (3) the organ of Corti in the cochlea. Centrally, the fine fibres of the cochlear nerve end in a ganglion in intimate relation with the restiform body, which may be described as consisting of two parts: (*a*) tuberculum acousticum, or lateral cochlear nucleus, and (*b*) ventral cochlear nucleus.

Central connections of cochlear nerves.—From the cells of these nuclei two tracts arise: a ventral tract, composed of the fibres of the corpus trapezoides, and a dorsal tract, represented by the striæ acousticæ. These tracts cross the middle line, decussating with the corresponding fibres of the opposite side, and then the strand turns upwards to become the lateral fillet: the fibres of the lateral fillet end in the inferior quadrigeminal body and the corpus geniculatum internum, and from the latter a tract proceeds to the cortex of the superior convolution of the temporal lobe. But it should be noted that certain internodes are situated in the corpus trapezoides and the lateral fillet, of which the most important is the superior olive, by means of which a communication is established with the nuclei of the third, fourth, and sixth nerves. In this way the organ of hearing is brought into connection with the nuclei which preside over the movements of the eyeballs.

The succession of neurons which build up the

sensory chain linking the organ of hearing to the cortex are therefore :

- (1) Bipolar cells of the ganglion spirale.
- (2) Neurons of the terminal cochlear nuclei.
- (3) Neurons of the superior olive and nucleus of lateral fillet.

- (4) Neurons of the corpus geniculatum internum.

2. The **vestibular nerve**, on which is the vestibular ganglion, enters the brain on the mesial side of the ventral cochlear nucleus; its fibres end in three nuclei of termination: (*a*) principal dorsal nucleus, (*b*) nucleus of Deiters, and (*c*) perhaps "nucleus of descending root."

Central connections of vestibular nerve.—Their connection with the nucleus of Deiters brings the fibres of the vestibular nerve into relationship with other parts of the mechanism of equilibration, for Deiters' nucleus constitutes an internode in the path of those fibres which connect the cortex and roof nuclei of the cerebellum with the motor apparatus of the spinal cord; it also receives fibres through the vestibular nerve from the labyrinth of the ear, and sends fibres into the posterior longitudinal bundle, through which, in all probability, it influences the nuclei of the ocular nerves.

Lesions of the auditory nerve.—The chief symptoms of disorder of the auditory nerve are deafness, tinnitus, and vertigo (Chapter XLVIII.). The nerve is very liable to injury, often in conjunction with the facial, from the pressure of tumours, especially those growing at the cerebello pontine angle, and it may also be damaged along with the facial in fractures of the base of the skull.

Atrophy of the auditory nerve sometimes occurs in locomotor ataxy.

VII.—THE GLOSSO-PHARYNGEAL AND VAGUS NERVES

Anatomical.—The motor or efferent fibres spring from the dorsal motor nucleus and the

nucleus ambiguus, lying deeply in the substance of the medulla; these fibres join the vagus and glosso-pharyngeal nerves.

The afferent fibres of the glosso-pharyngeal and vagus end in the dorsal nucleus and in the funiculus solitarius.

The glosso-pharyngeal nerve emerges from the upper part of the medulla oblongata, and leaves the cranium by the jugular foramen. It then appears in the interval between the internal jugular vein and the internal carotid artery, and is directed downwards and forwards to the tongue. It distributes branches to the mucous membrane of the tongue, pharynx, and middle ear, as well as to the stylo-pharyngeus and possibly to the middle constrictor of the pharynx.

The vagus, which also leaves the skull by the jugular foramen, is directed downwards in the cardiac sheath, and enters the thorax and abdomen. Each vagus is connected with the following cranial nerves: spinal accessory, glosso-pharyngeal, facial, and hypoglossal; also with some spinal nerves, and with the sympathetic in the neck, thorax, and abdomen.

Lesions of the glosso-pharyngeal.—The exact functions of the nerve are not known, owing to the rarity of cases of isolated paralysis, since it is intimately connected with the fibres of the vagus. Sense of taste may be impaired in the posterior third of the tongue, and there is some difficulty in swallowing, owing probably to paralysis of the stylo-pharyngeus.

Lesions of the vagus.—Affections of the nuclei in the medulla will be accompanied by unilateral and bilateral palsies, according to the extent of the disease. Lesions of one nerve cause unilateral paralysis of the soft palate and larynx, as is shown by the uvula being drawn over to the healthy side on phonation and by one vocal cord assuming a position nearly

midway between abduction and adduction—the so-called “cadaveric position.” The paralysed cord moves neither during phonation nor respiration, but, on phonating, the healthy cord moves over the middle line to meet it. Owing to their length, one or other vagus nerve is particularly likely to be damaged, and the left recurrent laryngeal, which winds round the arch of the aorta, is especially apt to suffer from pressure of aneurysms. In this case the paralysed cord assumes the position described above; the voice is hoarse and weaker than usual, the cough is harsh, and the power of coughing is much diminished owing to the inability to bring the cords firmly together.

The fibres to the abductors of the vocal cords are more easily damaged than those to the adductors; hence in the earlier stages, before the paralysis is complete, the unantagonised adductors are apt to draw the cord to the middle line (Semon) till they, in their turn, lose their function, and the cord swings into its position of complete paralysis. During the stage in which the abductor only is affected there are no definite symptoms to draw the patient's attention to his throat; the movements of the healthy cord are sufficient for ordinary respiration, and, since the two cords can meet in the middle line, vocalisation is not seriously interfered with. This stage of the paralysis is therefore likely to remain undiscovered unless a systematic examination of the larynx is made.

Both cords may be paralysed if the nuclei of the nerves are diseased, in which case the voice and the power to cough are both completely lost.

In paralysis of the abductors the cords come together in the middle line, and they do not separate during inspiration. The voice and power of coughing are but little affected, but there is

obstruction to respiration, which shows itself by loud stridor.

If the abductor is paralysed on *one* side only, the cord on that side will approach the middle line, but there will be no definite symptoms so long as the movements of the other cord are freely carried out. This, as already mentioned, is apt to occur from pressure on the recurrent laryngeal before the adductors are paralysed.

In adductor paralysis the cords cannot be brought together, and the voice is lost. This form of paralysis is usually only partial, and is the form which commonly occurs in hysteria, when the cords cannot be voluntarily brought together for phonation, but usually move freely for coughing, and therefore the patient may be able to cough although the voice is lost.

Spasm of the muscles of the larynx may occur from central or peripheral irritation.

The result of general laryngeal spasm is closure of the glottis owing to the predominating power of the adductors. One of the commonest forms of laryngeal spasm occurs in rickety children, under the name of *laryngismus stridulus*. The attacks, which occur chiefly at night, are characterised by acute dyspnœa, which lasts for some seconds, followed by loud crowing inspirations as relaxation of the muscles takes place.

Laryngeal spasms occur also in epilepsy and occasionally in hysteria. It may also be induced by local irritation, such as from foreign bodies or from tuberculous or syphilitic ulceration of the larynx.

The respiratory and cardiac rhythm may also be modified from disease of the vagus.

VIII. THE ELEVENTH NERVE: SPINAL ACCESSORY

Anatomical.—The spinal accessory nerve consists of two divisions. The *spinal division* arises

by a series of filaments from the cells of the anterior horns from the first to the fifth cervical segments, and passes upwards through the foramen magnum to join the accessory portion, in company with which it leaves the skull by the jugular foramen, and, after passing either in front or behind the internal jugular vein and perforating the sterno-mastoid, it crosses the posterior triangle of the neck, to end in the trapezius. It supplies this latter muscle as well as the sterno-mastoid.

The *accessory division* is to be regarded rather as an extension of the vagus. It arises from nuclei in continuation with those of the vagus, and after passing out of the skull with the spinal portion, leaves it to return again to the vagus, and it ultimately supplies fibres to the pharyngeal and superior laryngeal nerves.

The spinal accessory, like the vagus, is apt to be compressed by tumours and to have its nuclei affected in bulbar lesions.

The results of paralysis of the spinal portion are loss of power and wasting of the sterno-mastoid and upper part of the trapezius.

Paralysis of one sterno-mastoid gives rise to difficulty in rotating the head to the opposite side, and when power is lost in both there is a tendency for the head to fall back.

When the upper and middle fibres of the trapezius are paralysed the movements of shrugging the shoulders and raising the arm above the horizontal position are impaired, although the latter can still be performed by means of the serratus magnus.

When the lower fibres are paralysed the position of the scapula is altered, so that the vertebral border stands out prominently, as in paralysis of the serratus magnus, and there is also some loss of power to rotate the arm backwards.

The sterno-mastoid and trapezius often suffer together, but the nerve is sometimes affected after

it has left the sterno-mastoid, in which case the trapezius suffers alone.

The sterno-mastoid and trapezius receive fibres also from the second and from the third and fourth cervical nerves respectively.

IX.—THE TWELFTH, OR HYPOGLOSSAL NERVE

Anatomical.—This nerve arises from a nucleus situated in the floor of the fourth ventricle,

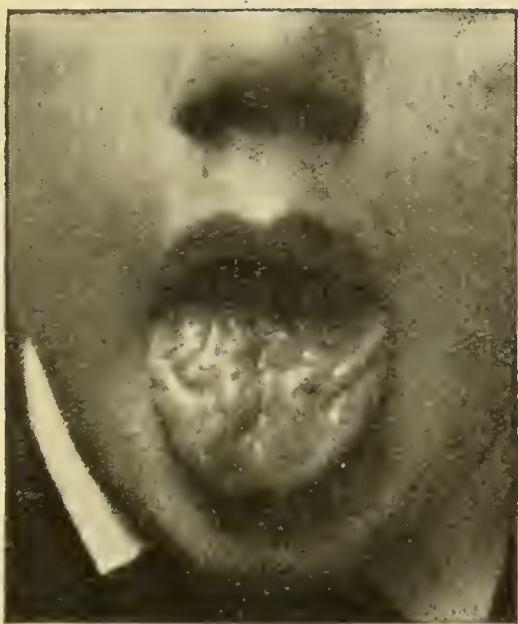


Fig. 12.—Atrophy of the tongue muscles.
Note the wrinkled surface.

and its filaments appear on the anterior surface of the medulla near the middle line, whence, uniting into a trunk, they pass through the anterior condyloid foramen.

The muscles chiefly affected in lesions of the hypoglossal are those of the tongue.

Lesions of the nucleus of the fibres of the nerve cause loss of power, perhaps at first only showing itself by indistinct pronunciation of certain letters, and later by paralysis, such that

the tongue cannot be put out or thrust into either check. As the muscle atrophies the mucous membrane becomes thrown into folds (Fig. 12).

When the muscles of one half of the organ are weakened the tongue, when protruded, will deviate towards the weakened side.

The nerve supply of the orbicularis oris, though running in the trunk of the facial, appears to come originally from the hypoglossal nucleus, and hence paralysis of the lips of nuclear origin is liable to accompany that of the tongue, as, for instance, often happens during the course of progressive bulbar paralysis.

The hypoglossal nucleus is especially liable to be the seat of degeneration in progressive bulbar paralysis; it may also be affected in syringo-myelia of the medullary region and by the pressure of tumours.

At the base of the skull, meningitis and tumours may compress the nerve trunks.

CHAPTER VII

PARALYSES OF THE UPPER LIMB AND OF THE CERVICAL SYMPATHETIC

THE UPPER LIMB

Anatomical.—The *brachial plexus* is made up of the fifth, sixth, seventh, and eighth cervical roots, together with the first dorsal root. The fibres of the roots enter into the composition of different nerve trunks, so that the distribution of the paralysis from a root lesion differs from that due to a lesion of a peripheral nerve.

It is thus necessary, for purposes of localisation, to distinguish between paralyses of root distribution and those of peripheral nerve origin.

The subjoined table is a copy of that compiled by Wilfred Harris* as the result of stimulation of nerves in operation cases, of observations on pathological conditions, and of dissections of the plexus. Harris points out that certain variations may be met with through plexuses being shifted either up or down. If the plexus is a high one (*i.e.*, prefixed) it receives a branch from the fourth cervical nerve with some curtailment of the branches from the lowest roots. If, on the other hand, the plexus is a low one (*i.e.*, postfixed) there will be no branch from the fourth cervical nerve, and the roots at the lower end will be of large size. These differences entail slight variations in the distributions of the roots. The table is from a prefixed type of plexus.

* "The True Form of the Brachial Plexus and its Motor Distribution," *Journ. of Anat. and Phys.*, July, 1904.

MOTOR ROOT SUPPLY OF MUSCLES OF UPPER
EXTREMITY. (*Wilfred Harris*)

PREFIXED TYPE OF PLEXUS

- 4-5 C. . . Deltoid.
Teres minor.
Supraspinatus.
Infraspinatus.
- 5 C. . . Rhomboids.
Subclavius.
Biceps.
Brachialis anticus.
Supinator longus.
Supinator brevis.
Pronator radii teres.
Extensor carpi radialis longior.
Extensor carpi radialis brevior.
- 5-6 C. . . Clavicular pectoral.
Subscapularis.
- 5-6-7 C. . Triceps.
Serratus magnus.
- 6-7 C. . . Teres major.
Coraco-brachialis.
- 6-7-8 C. . Pectoralis major (sternal).
Latissimus.
Extensor carpi ulnaris.
Flexor carpi radialis.
- 7-8 C. . . Flexor carpi ulnaris.
Pectoralis minor.
Extensor ossis metacarpi pollicis.
Extensor primi internodii pollicis.
Extensor communis digitorum.
- 8 C.-1 D. . Extensor secundi internodii pollicis.
Extensor indicis.
Extensor minimi digiti.
Pronator quadratus.
Palmaris longus.
Flexor sublimis digitorum.
Flexor longus pollicis.
Flexor profundis digitorum.
- 1 D. . . Thenar muscles.
Lumbricals.
Interossei.
Hypothenar muscles.

It is occasionally possible, in cases of injury of the cord, to analyse further the relative position

of the segments from which the muscles are innervated. I have recorded cases* from which it appears that the extensors and flexors of the wrist are situated at a higher level of the cord than the long flexors and extensors of the fingers and thumb, and, further, that the extensors of the wrist are represented at a higher level in the cord than the flexors of the wrist.

The motor and, of course, in many cases, the sensory roots are especially liable to suffer in injuries of the spinal column, *e.g.*, fractures and dislocations, and Farquhar Buzzard has shown that uniradicular palsies apparently depending upon a vascular lesion may sometimes occur. The whole or part of the brachial plexus may be severely injured by dislocations of the head of the humerus, or in elderly people it may be subject to inflammations which appear to be of a rheumatic or gouty nature.

The peripheral nerves may be crushed or severed, they may suffer as part of a more generalised neuritis, or they may be injured by continual pressure as from the use of crutches.

SUMMARY OF PARALYSES OF BRACHIAL PLEXUS

For purposes of classification, the paralyses of the brachial plexus may be grouped in three divisions, though, of course, they are often mixed.

1. Paralyses of the upper type, in which the loss of power corresponds to the distribution of the fifth cervical root.

2. Paralyses of the lower type, in which the weakness follows the supply of the first dorsal root.

3. Paralyses of the intermediate type, in which the fault lies in the sixth, seventh, and eighth cervical roots.

The fifth cervical root, owing to its anatomical position, is the one likely to suffer most when the

* *Brain*, Part lxxxv., 1899.

neck and shoulder are forcibly stretched apart. This sometimes happens to a child as it is being born, with the result that the deltoid, biceps, and supinator longus are paralysed. This condition is often known as "obstetrical paralysis." The arm is usually kept extended and rotated inwards by the unantagonised action of the triceps and other muscles. Whether recovery takes place or not depends upon the degree of primary injury, though happily in many cases the power of conductivity returns.

The line of treatment consists in preserving as far as possible the nutrition of the muscles by means of massage and electricity, until the functions of the nerve fibres are restored. If after some months there be no sign of any return of power, and no encouragement be obtained from the electrical reactions, the chances that the injured nerve will recover are very slight. In cases which show no signs of recovering the possibility of obtaining any improvement from an operative procedure by grafting the injured nerve on to a healthy one must be considered.

examples The eighth cervical and first dorsal roots are sometimes injured as they leave the cord by accessory ribs, tumours, or thickening of the meninges (Plate III.). This variety is known as lower plexus or Klumpke's paralysis. The small muscles of the hand waste and there is some pain or anæsthesia over the area supplied by the roots. In some cases the oculo-papillary fibres are implicated, with consequent myosis and narrowing of the palpebral fissure. The segments of the cord supplying the muscles of the hand are especially apt to be affected in progressive muscular atrophy and syringo-myelia.

In dislocations of the shoulder joint the circumflex nerve is very often stretched, and it is common, even after the dislocation has been reduced quite quickly, to find some weakness of the deltoid, as shown by inability to raise the arm

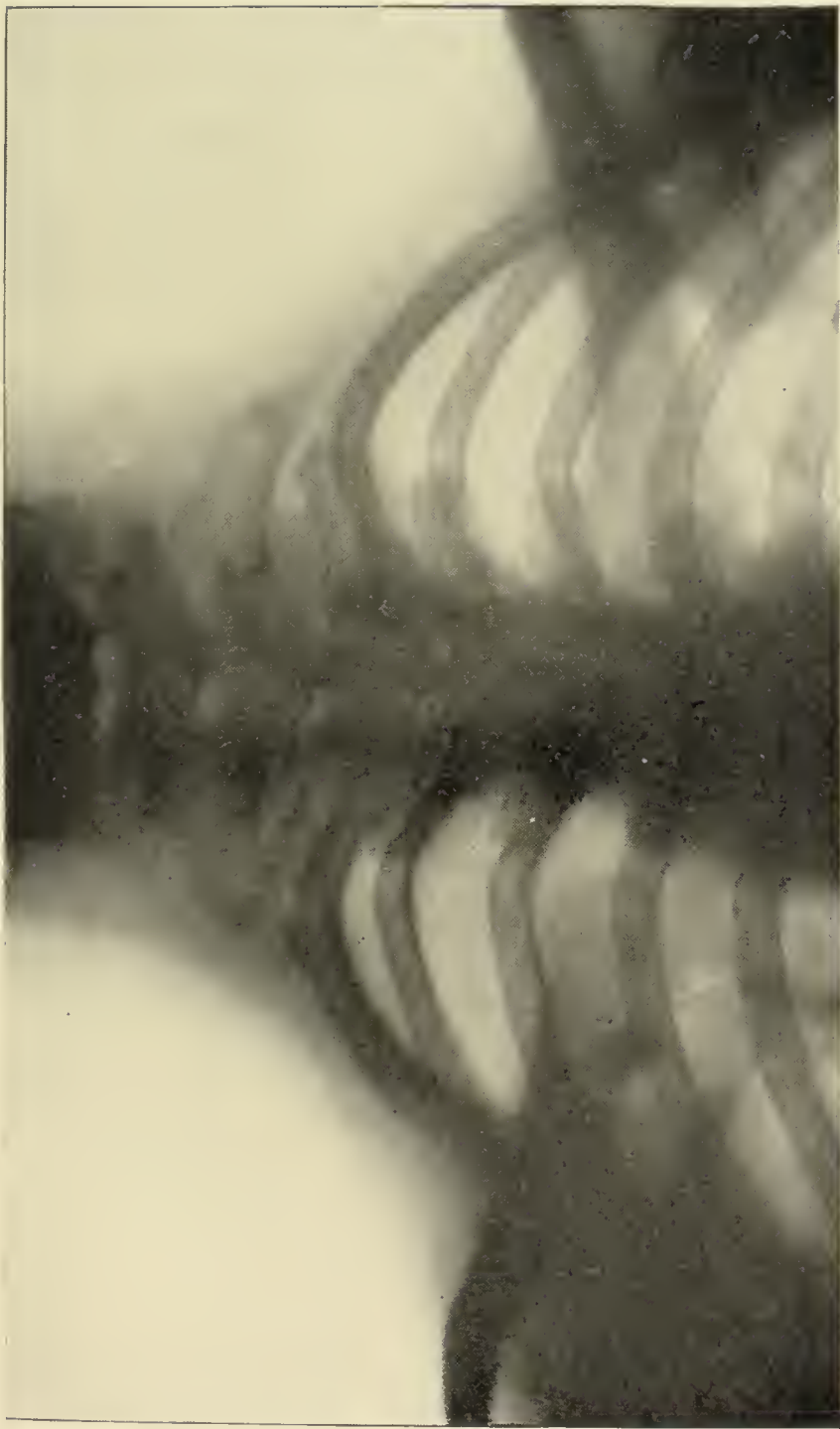


PLATE III.—Skiagram showing a Cervical Rib which gave rise to Symptoms of Pressure on the Lower Roots of the Brachial Plexus. (The rib was eventually removed by Mr. Pearce Gould.)



Fig. 13.—Paralysis of the deltoid. Note the position which the patient assumes in endeavouring to raise his arm.

to the horizontal position (Fig. 13). In other cases, especially those of subglenoid dislocation, the whole plexus may be severely contused, and if the head of the bone is not replaced within a reason-



Fig. 14. — Absence (congenital) of the pectoralis major on the right side. (*From a case under the care of Mr. T. H. Kellock.*)

able time the results may be disastrous. In such cases the arm is completely paralysed, the muscles waste rapidly, and the skin assumes the characteristic glossy appearance. There is usually agonising

pain, with varying degrees of anæsthesia, and often intense hyperæsthesia. Massage and electrical treatment must be given regularly, although at first, on account of the pain, both have often to be administered very lightly; voluntary movements must be strongly encouraged with the first sign of returning power.

For the pain, local sedatives are often useful, while, internally, phenacetin and similar drugs are beneficial. In many cases morphia is required, if only to produce the sleep that it may be otherwise impossible to obtain. In one case in which the pain was excruciating some relief was obtained by rapidly alternating applications of heat and cold.

Recovery in these severe cases is very slow, but may in the end be complete, provided methods of treatment are conscientiously carried out by the patient for a long time. One of the worst cases which I have had under my care got quite well after about three years.

The **pectoralis major**, which gets its nerve supply from the external and internal anterior thoracic nerves, is an important muscle to examine. It consists of an upper and lower division, and its action, which is chiefly to adduct the arm, can be shown by holding out the arms horizontally in front of the chest and then pressing them towards the middle line against resistance.

The lower division of this muscle is particularly liable to early atrophy in pseudo-hypertrophic paralysis; it is also sometimes absent from birth (Fig. 14).

LESIONS OF SPECIAL NERVES

The **long or posterior thoracic nerve** (nerve of Bell).—Injury or disease of this nerve results in paralysis of the serratus magnus. The chief functions of this important muscle are (1) to fix the scapula, so that other muscles may take

their action from it, and (2) to rotate the scapula (in conjunction with the trapezius) when the arm is raised above the shoulder.

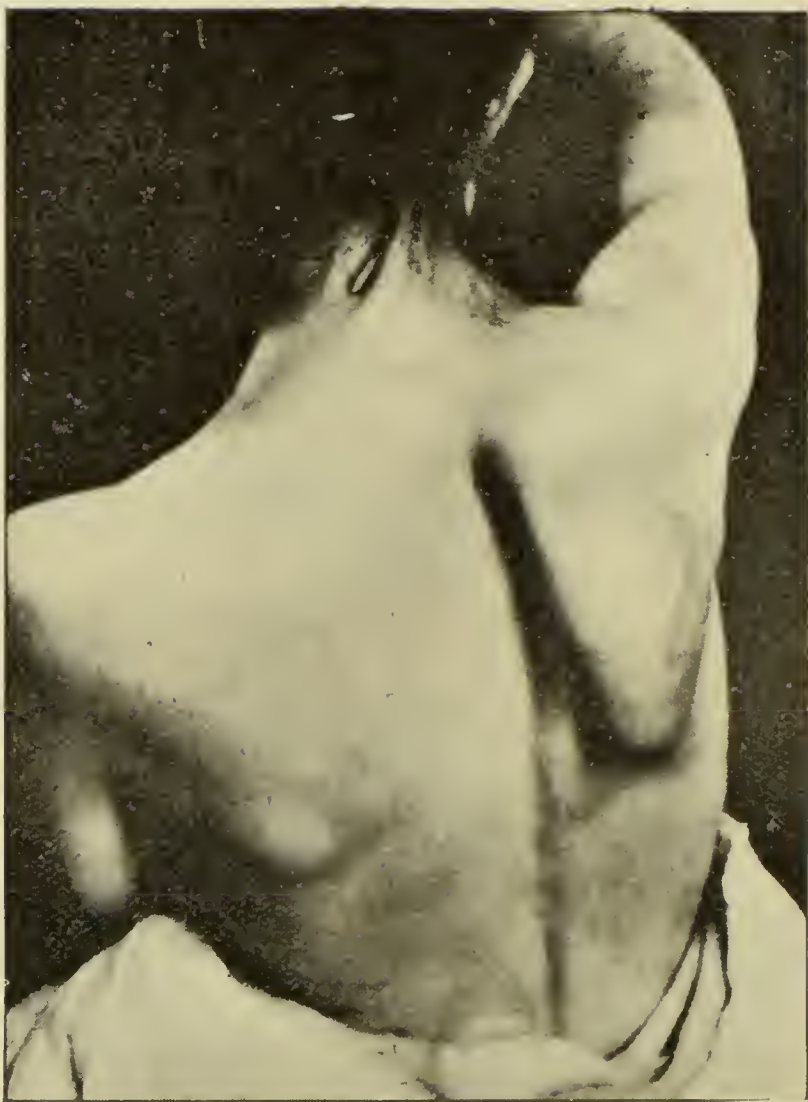


Fig. 15.—Paralysis of the serratus magnus. Note the “winging” of the vertebral border of the scapula when the arm is raised.

When the serratus is paralysed (Fig. 15) the scapula can no longer be fixed, and when the arm is raised it rotates so that its vertebral border projects and presents the wing-like appearance seen in the illustration. Owing to this want of fixation

the power of pushing is also greatly impaired. The diminished power of rotating the scapula is seen on attempting to raise the arm above the level of the shoulder, which movement, in the absence of the serratus magnus, is imperfectly performed by the trapezius. The effects of paralysis of the serratus magnus may sometimes be confused with those due to paralysis of the lower part of the trapezius. The vertebral border of the scapula stands out unduly in both, but, in the absence of serratus magnus paralysis, the deformity disappears when the scapula is rotated by raising the arm above the head.

There may also be some difficulty in deciding about the power of the serratus magnus when the deltoid is paralysed, so that the arm cannot be raised high enough to demonstrate the ability or inability to rotate the scapula. In these circumstances, Duchenne has shown that paralysis of the serratus magnus can be demonstrated by making a forward movement of the shoulder; if the muscle is acting, the lower angle of the scapula is moved forwards and outwards, while if it is paralysed, the spinal border does not move except to become rather more prominent through some rotation on its vertical axis.

Affections of the **long subscapular nerve** result in paralysis of the latissimus dorsi. Contraction of this muscle can be shown by adducting and depressing the arm against resistance.

C. E. Beevor* has pointed out that while one latissimus dorsi only acts in a voluntary adductor movement of the arm, the two muscles act together in such involuntary movements as coughing and sneezing, so that in cases of hemiplegia it may be possible to show loss of power of the muscle

* *Brit. Med. Journ.*, 1898, Vol. II., p. 976. As pointed out by Hughlings Jackson, the upper part of the trapezius acts in a similar manner; the highest fibres act unilaterally to elevate the shoulder and bilaterally when called into play to assist in respiration.

to perform voluntary movements; while those for involuntary ones are preserved, just as, in the face of a hemiplegic, the corner of the mouth will not move voluntarily, but will do so in the expression of emotions such as laughing and crying.

Beevor suggests that a knowledge of this double action may at times be useful in differentiating diseases of the brain from those of the cord and peripheral nerves: for whereas in cerebral lesions the involuntary bilateral movements might be preserved, in trouble at the periphery all movements would be lost.

Beevor has further shown that, though the latissimus has generally been considered to be a muscle of inspiration, its chief action is in reality expiratory in character.

The **musculo-spiral nerve**, the longest offshoot from the brachial plexus, is derived from the posterior cord. At first situated behind the third part of the axillary artery and the commencement of the brachial, it then winds round the back of the humerus in the musculo-spiral groove between the external and internal heads of the triceps, and appears upon the outer aspect of the arm, between the brachialis anticus on the inside, and the supinator longus and the extensor carpi radialis longior on the outside; opposite the external condyle of the humerus it divides into the radial nerve and the posterior interosseous. The musculo-spiral nerve supplies the three heads of the triceps, the supinator longus, the extensor carpi radialis longior, the brachialis anticus (in part), the anconeus and subanconeus.

The **posterior interosseous nerve** winds round the neck of the radius in the substance of the supinator brevis (which it supplies) to reach the back of the forearm, and then runs down between the superficial and deep extensors on the back of the forearm; these it innervates.

The **radial nerve** is purely sensory, and is

distributed to the dorsal aspect of the thumb as far as the bed of the nail, and to a varying extent of surface on the back of the index, middle, and ring fingers.

The musculo-spiral nerve may be involved in the callus formed round a fracture of the shaft of the humerus, especially where it is lying in the musculo-spiral groove. On the inner side of the arm it may be injured by the pressure of crutches or by the pressure induced by the arm hanging over a chair or other hard, resisting object, especially if the patient is under the influence of alcohol or other narcotic.

The fibres of the nerve, with the exception of those going to the supinator longus, are especially liable to be affected in chronic lead-poisoning. (*See* p. 109.)

Injury to the musculo-spiral causes weakness of the extensor muscles of the forearm and gives rise to wrist-drop, with which, according to the position of the lesion, there may be associated a paralysis of the triceps.

The **prognosis** of a paralysis from temporary pressure is usually favourable. A few days or a week generally suffices to restore power, but sometimes a much longer period of time is required, and recovery appears to be a distinctly slower process in elderly people.

The **ulnar nerve**, arising from the inner cord of the brachial plexus, runs along the inner side of the arm, and enters the forearm by passing between the internal condyle of the humerus and the olecranon. In the forearm it runs down upon the surface of the flexor profundus digitorum between the flexor carpi ulnaris on the inside and the flexor sublimis digitorum on the radial side; in the lower two-thirds of the forearm it is closely associated with the ulnar artery, lying upon the ulnar side of the vessel. It passes in front of the anterior annular ligament and divides into a

superficial and a deep division. In the forearm it innervates the flexor carpi ulnaris and the ulnar half of the flexor profundus digitorum, and it often gives off its dorsal cutaneous branch as high as the middle third of the forearm, which supplies the ulnar portion of the dorsum of the hand and the dorsal aspect of the inner one and a-half digits as far as the middle of the phalanges.

The nerve also supplies most of the intrinsic muscles of the palm, namely, the seven interossei, the two inner lumbricals, the three muscles of the



Fig. 16.—Ulnar paralysis. Note the hollow caused by the wasting of the first interosseous (abductor indicis) and the development of the “claw hand.”

hypothenar eminence, the two adductors of the thumb, and the deep head of the flexor brevis pollicis; it also supplies the skin over the palmar aspect of the inner one and a-half digits, sending branches on to the dorsum as far up as the middle and second phalanges.

The results of ulnar paralysis (Fig. 16) are loss of power, with wasting of the muscles mentioned above. Weakness of the interossei shows itself by inability to separate the fingers in the horizontal plane, and the wasting becomes obvious from the hollows between the metacarpal bones, especially the concavity of the radial side of the first metacarpal, due to loss of substance of the first dorsal inter-

osseous (the abductor indicis). The slight degree of abduction of the index and little fingers, which can still be performed by the extensors and lumbricals, must not be mistaken for the action of the interossei.

Loss of adduction, *i.e.*, the movement of the thumb which brings the ulnar border of the thumb alongside the metacarpal bone of the first finger, can be shown by asking the patient to press something between the ulnar border of the thumb and the first finger. In the absence or weakness of the adductor and inner head of the flexor brevis pollicis, the movement is feeble, and a hollow over the site of the muscles is visible. As the thumb moves from the position of abduction to that of adduction there is, in addition to the contraction of the adductors proper, also some accompanying contraction of the extensor longus pollicis and the flexor carpi ulnaris.^{*} In ulnar paralysis care must be exercised not to take movements produced in this way for those of true adduction. After some time the hand tends to assume the claw position (*main-en-griffe*), which must be distinguished from Volkmann's contracture (*see p. 89*). There is also anæsthesia over the area supplied by the nerve as detailed above.

It is important to remember that if the nerve be injured just above the wrist—the most frequent site of injury—the patient will retain sensation over the peripheral parts of the dorsal aspect of the ring and little fingers, while the proximal portions of these digits are quite anæsthetic. This is due to the fact that the dorsal cutaneous nerve is given off above the site of injury to the main trunk.

Again, abduction of the index and little fingers can be performed to a limited extent by their extensor tendons, and the contraction of the first lumbrical in the first space may be mistaken for

^{*} C. E. Beever, Croonian Lectures on Muscular Movements, 1904.

that of the abductor indicis. But if these fallacies are borne in mind no difficulty need arise in diagnosing a lesion of the nerve.

Being less exposed, the **median nerve** is less liable to injury than the ulnar, but may be damaged in fractures and dislocations of the humerus. It is most frequently injured just above the wrist by wounds, caused by such accidents as the bursting of bottles or thrusting the arm through a window.

The median nerve arises by two heads, one from the outer cord and the other from the inner cord of the brachial plexus. The nerve is first situated on the outer side of the axillary and the brachial arteries, but in the middle of the arm it crosses the latter vessel from without inwards, and runs down on the inner side as far as the elbow. It passes between the two heads of the pronator radii teres, being separated from the ulnar artery by the deep head of the latter muscle, and then continues straight down the forearm between the flexor sublimis and flexor profundus digitorum muscles; just above the anterior annular ligament, it lies exactly posterior to the palmaris longus tendon, if this is present. It passes behind the anterior annular ligament enveloped by the synovial sheath which is wrapped round the tendons of the flexors of the fingers, and, reaching the palm, divides to supply the muscles of the thenar eminence, viz., the abductor pollicis, opponens pollicis, and superficial head of the flexor brevis pollicis. It innervates the two outer lumbricals, and supplies the skin over the palmar aspect of the radial side of the hand, over the front of the thumb, the index, middle, and half the ring fingers, and over varying portions of the dorsum of the same.

In the forearm the median supplies all the muscles arising from the internal condyle, except the flexor carpi ulnaris, and through its anterior

interosseous branch it innervates the deep muscles of the forearm, viz.: the flexor longus pollicis, the pronator quadratus, and the outer half of the flexor profundus digitorum (the inner half of the latter muscle deriving its nerve supply from the ulnar nerve).

In cases of lesion of the median nerve there will be a loss of power in the muscles innervated by the nerve, and anæsthesia over the area of skin which it supplies; but the action of some of the muscles to which the median is distributed may be closely simulated by others drawing their nerve supply from a different source. It may be impossible to tell from inspection whether a particular movement is produced by the opponens pollicis or by the adductors and flexors of the thumb. Palpation over the insertion of the former muscle into the metacarpal of the thumb is the only way to avoid errors in these cases. (Sherren.)

Paralysis of the two outer lumbricals is masked by the action of the interossei. (Sherren.)

Beever has pointed out that true abduction of the thumb takes place in a plane at right angles to that of the palm. This is the action of the abductor pollicis, and is but feebly imitated by the extensor ossis metacarpi pollicis, when some extension accompanies the movement.

VOLKMANN'S CONTRACTURE, OR ISCHÆMIC PARALYSIS

This form of paralysis was first described by Volkman in 1875, and is characterised by a contraction of the forearm muscles, which causes contraction of the fingers and sometimes also of the wrist.

Etiology.—The affection usually arises during treatment for fracture in the region of the elbow-joint, especially in connection with fractures of the lower end of the humerus, or a separation of the lower epiphysis of that bone. Undue pressure by splints and tight bandaging appears to play the

most important part in the production of the contracture, as is evidenced by the fact that in a large number of the cases the splints exercised sufficient pressure to cause sloughing. It is not so much that the splint is applied too tightly at first as that no allowance is made for the swelling that is certain to ensue in the next few days.

Prolonged obstruction to the circulation by an Esmarch's bandage, injury to the brachial artery, rupture or severe contusion of the flexors, and prolonged suppuration in the muscles have also been assigned as causes of the contracture.

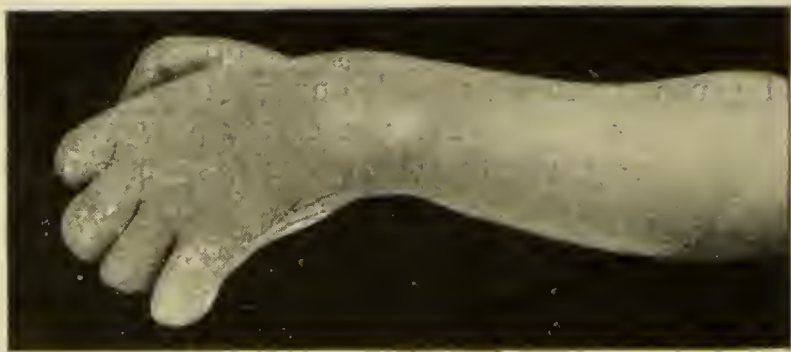


Fig. 17.—Position of hand in an early stage of ischemic paralysis.

The condition is most frequently met with in children.

Symptoms.—The chief characteristics are hardness of the muscles, followed by contraction of the flexors of the forearm, causing flexion of the fingers, and sometimes of the wrist; and in severe cases the forearm is fully pronated and fixed in this position. When the wrist is flexed it may be possible to uncoil the fingers to a varying degree, but as soon as the wrist is extended, the pull of the tightened flexors is felt, and the hand again assumes a claw-like aspect, the metacarpophalangeal joints being in a condition of hyperextension and the middle and terminal phalanges clenched in extreme flexion (Figs. 17 and 18).

Pathology.—The exact nature of the condition is not quite clear, and the term "ischæmic" has been applied on the supposition that the pressure of the splints induces the changes by arresting the circulation in the muscles. The rapidity with which the symptoms show themselves, together with the fact that in some cases



Fig 18.—Position of fingers caused by contraction of the flexor tendons in ischæmic paralysis.

there are no alterations in the electrical reactions and no anæsthesia, supports the hypothesis that the lesion is primarily one of muscular rather than of nervous origin. The pale, firm, fibroid condition of the muscles is also in favour of this view, and the very early onset of the contracture in some cases, supervening even in a few hours, suggests the presence of some coagulation process or local "rigor mortis" of the compressed fibres as the pathological condition rather than a myositis,

though this very early onset may possibly be due to nerve spasm. On the whole, an inflammatory condition of the muscular fibres seems the most likely cause of the contracture.

In some cases, electrical changes and anæsthesia have been observed, but this fact does not necessarily invalidate the hypothesis that the disease is of muscular origin, for it is quite possible for injuries to the nerves to co-exist with Volkmann's contracture, and sometimes the median and ulnar nerves have been found imbedded in scar tissue, but otherwise healthy in appearance.

Diagnosis.—The diagnosis is based upon the history, the character of the deformity, the absence of electrical changes, the frequent absence of anæsthesia, and the simultaneous onset of paralysis and contracture.

Ulnar paralysis, in its later stages, to a certain extent resembles Volkmann's contracture, but in the former there is no flexion of the wrist, while the small muscles of the hand (except those supplied by the median) are wasted and show the reaction of degeneration. Further, there may be anæsthesia or paræsthesia over the area of skin supplied by the ulnar nerve.

Late musculo-spiral paralysis.—In this condition the extensors of the wrist and fingers are paralysed and give the reaction of degeneration. The supinator longus and triceps may share in the paralysis, and the terminal phalanges can still be extended by the interossei and lumbricales.

Infantile paralysis is distinguished from Volkmann's contracture by the history, the presence of the reaction of degeneration, and the fact that contracture follows the paralysis at a later date.

Treatment.—Massage, passive movements, and electricity may lead to much improvement in mild cases, but they must be commenced early and practised regularly. Even in the severe cases, although these measures fail to cure the deformity,

they are nevertheless of value in maintaining the nutrition of the muscles, and should be given a fair trial for three or four months before recourse is had to operative treatment. Operative measures include (1) tendon-lengthening and (2) excision of portions of the radius and ulna.

The latter method was first suggested by Raymond Johnson, and the following advantages over lengthening the tendons are claimed for it by Rowlands, who has also made use of it: (a) the operation is easier and takes less time; (b) the radius can be divided and sutured in such a manner as to increase the power of supination of the hand, which is practically always limited; (c) there is no interference with the tendons, and therefore no risk of non-union or weakness of any of these tendons; (d) if any contraction of the flexors of the wrist be present, this is also corrected at the same time by this single operation; (e) no nerves are liable to injury in this operation.

It has been objected that the bones may fail to unite, but, if carefully wired, non-union should not occur.

Rowlands shortens the forearm about three-quarters of an inch, and states that the best time for operation is possibly about four or five months after the date of onset.

Prognosis.—Some mild cases may recover by the systematic employment of active and passive movements, massage, and electricity, but in many cases some operative measure is necessary to permit of a more extensive range of movement for the diseased muscles and to correct the deformity before the former method of treatment can be of any permanent value.

THE CERVICAL SYMPATHETIC SYSTEM

Anatomical.—The cervical sympathetic system supplies (1) motor fibres to the dilator muscle of the iris, to the unstriated muscle fibres in the

upper eyelids, and to Müller's muscle, which consists of a small collection of unstriated fibres that pass across the spheno-maxillary fissure at the back of the orbit; (2) secretory fibres to the sweat glands of the head and neck and to the sub-maxillary salivary glands; (3) vasomotor fibres to the vessels of the head and neck.

The fibres which influence dilatation of the pupils arise in the medulla and pass down the spinal cord to the ciliospinal centre, which is situated in the cervical region. They leave the



Fig. 19.—Paralysis of the cervical sympathetic of the right side. Note the ptosis and enophthalmos of the right eye; also the pupil is smaller.

cord in company with the anterior roots of the first three dorsal nerves, and, after passing up the cervical sympathetic to the Gasserian ganglion, they reach the pupil by the long ciliary branches of the fifth nerve.

It will be seen from this that the dilator fibres may be injured by diseases and injuries before they leave the cord.

The vasomotor fibres appear to leave the cord at the level of the third to sixth dorsal nerves.

Etiology.—Paralysis of the cervical sympathetic system may arise from gunshot wounds, stabs, enlarged glands, tumours, and injuries to

the neck of any other description. The fibres appear sometimes to be involved in the fibrosis at the apex of the lungs which occurs in consequence of tuberculosis.

Symptoms.—(a) Ptosis.—The palpebral fissure is narrowed from paralysis of the unstriped muscle in the levator palpebræ, which causes the lid to droop (Fig. 19).

(b) Enophthalmos.—Paralysis of Müller's muscle is followed by some gradual retraction of the eyeball.

(c) Pupil.—The pupil on the affected side is contracted from the unantagonised action of the sphincter muscle, which is supplied by the third nerve, and it no longer dilates when it is shaded or when the cervical sympathetic is stimulated in the neck, but, owing to the integrity of the third nerve, it still contracts to light and during accommodation.

(d) Vasomotor symptoms.—Dilatation of cutaneous vessels on the paralysed side of the head and face sometimes occurs.

(e) Anidrosis.—Absence of sweating on the side of the paralysis may be present, but it is not a constant symptom, and hyperidrosis sometimes occurs.

If the cervical sympathetic is irritated the signs are reversed, but symptoms of irritation are rarely met with in comparison with those of paralysis.

CHAPTER VIII

PARALYSES OF THE LOWER LIMB

THE lumbar and sacral plexuses, like the brachial, may suffer from pressure, and the effects are likewise variously distributed according as the pressure affects the roots or the peripheral nerves.

The following table, taken from Morris's Anatomy, shows the relations of the muscles to the roots of the lumbar and sacral plexuses:—

NERVE ROOTS	MUSCLES
2 and 3 L.	<u>Ilio-psoas</u> . <u>Sartorius</u> . <u>Pectineus</u> . <u>Adductor longus</u> .
2, 3, and 4 L.	<u>Gracilis</u> . <u>Adductor brevis</u> .
3 and 4 L.	<u>Quadriceps extensor</u> . <u>Obturator externus</u> .
3, 4, and 5 L.	<u>Adductor magnus</u> .
4, 5 L. and 1 S.	<u>Gluteus medius</u> . <u>Gluteus minimus</u> . <u>Tensor fasc. femoris</u> . <u>Seminembranosus</u> . <u>Plantaris</u> . <u>Popliteus</u> . <u>Quadratus femoris</u> . <u>Inferior gemellus</u> .
5 L. and 1 S.	<u>Flexor long. digit</u> . <u>Tibialis posticus</u> . <u>Flexor brev. digit</u> . <u>Flexor brev. hallucis</u> . <u>Abductor hallucis</u> . <u>First lumbrical</u> .

NERVE ROOTS	MUSCLES
5 L., 1 and 2 S. . . .	Superior gemellus. Obturator internus. Gluteus maximus. Semitendinosus. Soleus. Flexor long. hallucis.
1 and 2 S.	Pyriformis. Gastrocnemius. Flexor accessorius. Abd. min. digiti. Plantar interossei. Dorsal interossei. Adductor hallucis trans. Adductor hallucis obliq.
1, 2, and 3 S.	Long head of biceps. Ext. long. hallucis. Ext. long. digiti. Ext. brev. digiti.
4, 5 L. and 1 S. . . .	Tibialis anticus. Peroneus tertius, longus, brevis.

The ilio-inguinal and ilio-hypogastric nerves arise from the first lumbar root, and, together with the genito-crural nerve, which arises from the first and second roots, terminate by supplying the skin over the upper part of the thigh.

The obturator nerve arises from the second, third, and fourth lumbar nerves. From its branches the adductor longus, adductor brevis, adductor magnus, gracilis, obturator externus and pectineus derive their nerve supply.

When this nerve is paralysed there is partial loss of power to adduct the thigh. The adductor magnus still retains some power, since it is partially supplied from the great sciatic nerve, and adduction may also be assisted by the pectineus, which often derives some of its supply from an accessory obturator nerve.

The external cutaneous nerve arises from the second and third lumbar roots. It supplies

the skin over the outer part of the thigh with sensation. Tingling and paræsthesia of various kinds are apt to occur in the distribution of this nerve.

The **anterior crural nerve** springs from the second, third, and fourth lumbar roots, which unite to form one trunk between the psoas and iliacus muscles. Besides sending branches to the iliacus, pectineus, and sartorius, it also supplies the quadriceps extensor, which is composed of the rectus, vasti, and crureus. The middle and internal cutaneous nerves, which supply the skin over the lower two-thirds of the anterior and inner surfaces of the thigh, and the long saphenous nerve, which supplies sensation over the anterior and inner part of the leg, all arise from the anterior crural.

The chief motor result of disease of the anterior crural nerve is loss of power to extend the knee, from paralysis of the quadriceps extensor. The knee-jerk is diminished or lost. Flexion of the hip is weakened through paralysis of the iliacus, but it can still be carried out by the psoas, which is supplied by special branches from the second and third lumbar nerves.

Even when all power of extending the knee-joint is lost, it may still be possible to stand and walk, through the patient learning to keep the leg in such positions that the extensors are not required.

The **superior gluteal nerve** arises from the lumbo-sacral cord and the first sacral nerve. It is responsible for the nerve supply of the gluteus medius and minimus, and the tensor vaginæ femoris.

The **inferior gluteal nerve** supplies the gluteus maximus. The gluteus maximus is one of the principal extensors of the thigh, but is also an abductor and outward rotator. Its action can be tested by asking the patient to lie flat on his face and then to raise his thigh. The gluteus

maximus is also of importance from the frequency with which it is enlarged in pseudo-hypertrophic paralysis. The gluteus medius and minimus are both abductors, but they also to some extent flex and rotate the thigh inwards.

The **small sciatic nerve** arises from the second and third sacral roots. After running down the back of the thigh and giving off branches which supply the skin over the back of the thigh and popliteal space, it pierces the deep fascia of the leg below the knee and supplies the skin over the upper part of the calf.

GREAT SCIATIC NERVE

The great sciatic nerve arises from the sacral plexus at the lower border of the great sacro-sciatic foramen. It runs down the back of the thigh and terminates by dividing into the external and internal popliteal nerves.

As the great sciatic nerve passes down the thigh, branches leave it to supply the biceps femoris, semimembranosus, semitendinosus, and a portion of the adductor magnus, which may all suffer in severe cases of sciatica.

At the top of the popliteal space the main trunk divides into external and internal popliteal nerves.

The **external popliteal nerve** (peroneal nerve) descends obliquely along the outer side of the popliteal space, running close to the biceps tendon. It then winds round the neck of the fibula (in which position it is very liable to injury), and, passing between the latter and the peroneus longus, it divides into the **anterior tibial** and the **musculo-cutaneous nerves**.

The external popliteal nerve may be injured in punctured wounds of the upper part of the popliteal space or at the neck of the fibula, and also when the biceps tendon is divided subcutaneously.

The anterior tibial nerve, commencing between the fibula and the peroneus longus, inclines obliquely beneath the extensor longus digitorum to reach the anterior surface of the interosseous membrane. It runs down the front of the limb till it reaches the level of the ankle joint, where it divides into internal and external branches, the former supplying the integument on the neighbouring sides of the great toe and the second toe on their dorsal aspect, the latter being distributed partly as motor branch to the extensor brevis digitorum, and partly as sensory twigs to the dorsal interossei. In the leg, the anterior tibial supplies the tibialis anticus, extensor proprius hallucis, extensor longus digitorum, and peroneus tertius.

Paralysis of the anterior tibial nerve leads to "foot-drop." The foot cannot be flexed on the leg, and the difficulty in walking, consequent on the toes catching in the ground, gives rise to a "high-stepping" gait.

The musculo-cutaneous nerve, lying in a sheath in the intermuscular septum between the peronei externally and the extensor longus digitorum internally, runs down in front of the fibula to the lower third of the leg, where it pierces the deep fascia in two branches, internal and external. The main trunk innervates the peroneus longus and brevis, and of the two terminals the internal branch comes down over the anterior annular ligament and supplies the lower third of the leg, the skin over the dorsum of the foot, the inner side of the big toe, and the adjacent side of the second and third toes. The external branch supplies the adjacent sides of the third and fourth and fourth and fifth toes, communicating with the external saphenous nerve. The action of the peroneus longus and brevis is to abduct the foot and also to aid in extending the ankle joint.

When these muscles are paralysed the power of abduction is lost, and the foot is turned inwards

by the unantagonised action of the tibialis anticus and posticus.

The internal popliteal passes onwards through the popliteal space, and at the lower border of the popliteus becomes continuous with the posterior tibial nerve. The latter runs down the back of the leg and terminates in the internal and external plantar nerves. The internal popliteal and posterior tibial nerves supply the gastrocnemius, soleus, plantaris, popliteus, tibialis posticus, flexor longus digitorum, and the flexor longus hallucis.

In paralysis of these muscles there is loss of power to extend the foot on the ankle joint, and to flex the toes. In long-standing cases the unantagonised action of the flexors produces a condition of talipes calcaneus, in which only the heel can be placed on the ground.

The internal plantar nerve is mainly sensory, supplying the palmar surface of the inner three and a half digits, but also innervating four muscles, *i.e.*, abductor hallucis, flexor brevis hallucis, flexor brevis digitorum, and the first lumbrical. The external plantar supplies the remaining sensory area and all the other muscles of the foot. As the digital branches from the plantar nerves run forwards to supply the skin covering the toes, they pass between the heads of the metatarsal bones, and, as a result of the crowding together of the bones by pressure of tight boots, the nerves going to supply adjacent sides of the fourth and fifth toes may be compressed. Pain may not only be felt at the actual site of pressure, but may radiate up nearly as high as the knee.

SCIATICA

Sciatica is one of the most common of the nervous diseases.

The causes may be divided into (1) extrinsic, such as pressure from pelvic tumours and spicules

of bone, and (2) intrinsic, depending upon inflammatory processes in the nerve trunk. These are often brought about by rheumatic and gouty conditions, and in some cases lead may be a factor. Injury, strain, damp, and cold all also appear to be causes.

Pathology.—An interstitial inflammation of the nerve sheath with subsequent injury to the nerve fibres is the most common lesion.

Symptoms.—The main symptoms are pain and tenderness. The pain is distributed in the course of the nerve in the thigh, and is often continued down the branches of the nerve beyond. There may be hyperæsthesia, and in some cases some degree of anæsthesia. In severe cases tenderness may be elicited by deep pressure anywhere along the line of the nerve, and it is nearly always present at certain points, *e.g.*, the sciatic notch, the popliteal space, and at the outer side of the fibula, at which places the nerve is more readily reached.

The pain is often very intense, and may prevent the patient from sleeping. The position of greatest ease is obtained by flexing the leg at the knee and hip, and any attempt to extend the leg at once stretches the nerve and causes pain. The position in which the leg is kept causes the patient to stand and walk in such a manner that the weight is chiefly thrown on to the opposite limb, and in time this attitude may cause some lateral curvature of the spine.

In severe and long-standing cases there may be atrophy of muscles, but in the recent and slighter cases there is often no definite loss of power. Occasionally œdema of the leg and herpes are present. The tendo Achillis jerk is usually lost on the affected side; the knee-jerk generally remains, and may be somewhat exaggerated. Lumbago is often associated with sciatica.

Diagnosis.—The diagnosis of sciatica is im-

portant, since it is necessary to be careful not to overlook possible causes of pressure. The pelvis must be examined for signs of tumour, and the spine for signs of tuberculosis, with which a psoas abscess may be associated. Account must also be taken of any injury or disease of the pelvic bones and hip joint, the effects of which may have extended to the sciatic nerve.

The lightning pains of tabes sometimes settle in the course of the sciatic nerve for days at a time. Unless this possibility is remembered, their nature is likely to be overlooked. Suspicion should always be aroused if the "sciatica" is bilateral.

Treatment.—At the outset of an acute attack the patient should be kept in bed and given a purge. To relieve the pain, all the ordinary forms of counter-irritation can be tried, and of these heat often proves the most beneficial, while phenacetin and drugs of similar nature may be given by the mouth. Where the pain is very severe, and not relieved by other measures, morphia must be used. Applications of the galvanic current may also help to relieve the pain.

As soon as the more acute symptoms have subsided, massage and electricity may often be prescribed with success; indeed, massage is one of the most important remedies we have. It must generally be light at first, though occasionally patients obtain considerable after-benefit from kneading in the acute stages, which at the time has given rise to a good deal of pain. In the more chronic cases applications of the high-frequency current are often useful.

Prognosis.—The prognosis varies. Some patients recover rapidly, while in others the disease becomes chronic and lasts for weeks or months. There is a considerable tendency towards recurrence.

CHAPTER IX

MULTIPLE NEURITIS

IN multiple or peripheral neuritis there is a generalised inflammation of some or all of the fibres forming nerve trunks. The nerves of the limbs are mainly affected. The symptoms are generally symmetrical, but vary according to the acuteness of the onset and the selective action which different poisons tend to exert.

Selective influence of poisons. — Many poisons which attack the nervous system have an apparent affinity for certain neurons before they produce more generalised symptoms, and a knowledge of their special tendencies is often an aid to diagnosis.

Thus, lead shows a special tendency to affect the nerves of the forearms, alcohol those of the legs, and diphtheria those of the pharynx and eyes.

Etiology.—Of the poisons taken directly into the system, those which most frequently produce neuritis are alcohol, lead, and arsenic, and less frequently copper and mercury. Toxins, which are the direct outcome of bacillary infection, are also causes, e.g., those associated with diphtheria, typhoid, malaria, pneumonia, tubercle, and other infectious diseases. Of these the diphtheria toxin is by far the most important, and, indeed, peripheral neuritis following the others is distinctly uncommon.

The toxins associated with leprosy and beriberi

poisons.

alcohol
arsenic

mercury

Toxins

Diphtheria

Influenza

Typhoid

Malaria

Pneumonia

Leprosy

uncommon

are also potent causes in countries where these diseases are prevalent.

Other poisons, probably of chemical rather than bacillary origin, which may cause neuritis are those of *gout, rheumatism, anemia, and diabetes.* Neuritis may also follow injuries and the general enfeeblement that is associated with wasting diseases and senile degeneration.

gout
Rheumatism
Anemia
diabetes

Pathology.—The changes found in the nerves in multiple neuritis are those of a parenchymatous inflammation and degeneration, to which may be added more diffuse inflammatory changes in the interstitial tissue and nerve sheaths, according to the nature and virulence of the poison. As a general rule the changes are most advanced in the terminal filaments.

In many instances, some changes have been observed in the motor cells of the cord also, thus showing that the poison has a tendency to attack the whole of the lower motor neuron.

General symptomatology.—An ordinary “mixed” nerve contains motor and sensory fibres, and possibly some fibres which exercise a trophic influence, although the independent existence of these is more than doubtful. A peripheral nerve may thus be divided into its various component fibres, like the columns of the cord, and, like the latter, all the parts need not be affected equally.

When the functions of all the fibres are impaired or destroyed, there is loss of power, loss of sensation, and loss of reflexes, with perhaps some trophic changes in the skin, which last are not, however, of such an acute nature as are found in myelitis.

The loss of power is mainly in the limbs, and may be confined to the legs or to the arms, or may be present in both. The weakness is usually most marked in the extensor muscles below the knees and elbows, giving rise to “dropped foot” and “dropped wrist.” The trunk muscles and those

supplied by the cranial nerves are very seldom affected, but the pneumogastric and phrenic nerves are especially liable to involvement in post-diphtheritic neuritis.

The signs are those of a lower neuron lesion, viz., wasting and alterations in the electrical reactions of muscles, loss of reflexes, and a tendency for the limbs to assume abnormal positions. The rapidity with which power is lost varies greatly, and in some of the acute toxæmic cases the limbs may become powerless in even a few hours. The knee-jerks are generally lost early, but occasionally they are temporarily exaggerated during the period of nerve irritation.

Sensory symptoms are often among the earliest to appear. Apart from complete anæsthesia, irritation of the sensory fibres can cause every variety of sensory disturbance, including pain, tingling, numbness, and hyperæsthesia of different intensities. When definite anæsthesia develops, it is usually symmetrical in the limbs, and corresponds to the areas over which gloves and stockings are usually worn, and hence it has received the name of "glove-and-stocking anæsthesia." The anæsthesia does not extend to the trunk. The muscles of the legs are often very tender, especially in the alcoholic cases, and in some instances loss of muscle-sense, with consequent incoordination, is one of the most prominent symptoms. The intensity and rapidity of the onset of sensory symptoms are very variable: in most of the chronic cases the subjective sensations of tingling, numbness, and pins-and-needles often come on gradually and are the first symptoms to which attention is drawn. The extent to which the different forms of sensation are impaired is also very variable: in some patients all forms are affected; in others the loss of muscle-sense may be disproportionately great, with a resulting ataxy that may be mistaken for that of tabes. In some of the more acute

varieties of toxic origin, sensations may be but little affected.

Vasomotor and trophic symptoms are not, as a rule, prominent. Œdema of the legs may be occasionally observed. The "trophic" changes show themselves chiefly by impaired nutrition of the skin, which becomes smooth and glossy; of the nails, which become brittle and lined; and of the smaller joints, which are apt to become stiff and to contract adhesions. Acute bedsores do not arise, and control over the sphincters is retained.

ALCOHOLIC NEURITIS

Etiology.—Alcoholic neuritis occurs from excessive drinking over a long period of time. Spirits appear to be the more potent cause, but many cases arise from drinking beer. Both men and women are affected.

Symptoms.—The loss of power nearly always begins in the feet and legs, and is most marked below the knees. The feet drop into a line with the legs (foot-drop), and if the patient is able to walk the gait is high-stepping in order to clear the toes from the ground. The arms are sometimes affected, and palsies of cranial nerves have occasionally been observed. The wasting of the paralysed limbs is generally very marked, and the electrical reactions are those of degeneration. The knee-jerks and plantar reflexes are usually lost.

The **sensory symptoms** are prominent. Thus pain is often excruciating, and the muscles of the calves may be very tender to pressure, although the latter sign is by no means always present. Numbness and tingling are common, as also is hyperæsthesia. The anæsthesia takes the characteristic "stocking-and-glove" distribution already described. The muscle-sense is apt to be greatly diminished, giving rise to ataxy resembling that of tabes.

The vision occasionally suffers; there may be

amblyopia or impairment of colour vision, and optic neuritis has been known to occur, though very rarely.

Mental symptoms.—Emotional tendencies, untruthfulness, indecision, mental confusion, with loss of memory for recent events and loss of ideas of space and time—symptoms which are grouped under the heading of *Korsakow's psychosis*—are generally met with in some degree, while in the more severe cases hallucinations, delirium tremens, or insanity may be present. These mental changes are sometimes associated with loss of control over the sphincters, a symptom which is not present in uncomplicated cases of multiple neuritis.

Pathology.—The chief changes are parenchymatous inflammation and degeneration of nerve fibres, the terminal twigs suffering most severely. The cells of the cord and cortex may also be altered, and it is to changes in the latter that the cerebral symptoms are due.

ARSENICAL NEURITIS

Etiology.—Arsenical poisoning has occurred from the inhalation of particles arising from various decorative articles into the composition of which arsenic has entered, as green wall-papers and carpets. Arsenic may also accidentally contaminate articles of diet in sufficient quantities to cause symptoms, as occurred in an epidemic among beer-drinkers due to the presence of arsenic in glucose. Neuritis may also be caused by long-continued use of arsenic as a medicine.

Symptoms.—There is a close similarity between the symptoms of arsenical neuritis and those of alcoholic neuritis, to which the reader is referred.

The legs are generally affected first (Fig. 20), and pain, tingling, and numbness are accompanied by weakness of the extensors and by ataxy. Later on the paralysis may appear in the arms.

Arsenical neuritis is accompanied by pigmentation of the skin, which is a symptom of diagnostic importance. The skin assumes a deep brownish colour, which is most apparent on the trunk and extensor surfaces of the limbs. Herpes, keratosis of the palms of the hands and soles of the feet, erythema, erythromelalgia, changes in the structure of the nails, and œdema of the extremities may all be present. Recovery generally takes place very slowly



Fig. 20.—“Foot-drop” in arsenical neuritis. (*From a case under the care of Dr. George Ogilvie.*)

LEAD NEURITIS

Etiology.—Neuritis from lead is chiefly found in painters, who inhale the poison, and who, unless they exercise great care, also contaminate their food with particles that have clung round the finger-nails. Workers with metallic lead, such as type-setters, are also liable to the disease. Another source of poisoning is the contamination of water or other drinking fluid with lead.

Symptoms.—Neuritis does not usually occur until the patient has been exposed to the influence

of lead for a long time, and it is generally preceded by other indications of lead poisoning, such as colic, dyspepsia, constipation, and anæmia.

The characteristic blue line on the gums can also often be observed, and analysis of the urine and fæces may afford confirmatory evidence of the



Fig. 21.—“Wrist-drop” in neuritis from chronic lead-poisoning.

presence of lead in the body. The inflammatory and degenerative changes are chiefly found in the peripheral nerves, but lesions can also occasionally be demonstrated in the motor cells of the cord.

The extensor muscles of the forearms and fingers are weakened, causing wrist-drop and inability to straighten the fingers (Fig. 21).

The supinator longus escapes, the paralysis being usually limited to the distribution of the posterior interosseous branch of the musculo-spiral

nerve. Though the flexors of the forearm are not paralysed, the grasp is greatly weakened owing to the inability of the extensors to fix the wrist. One hand is often affected before the other, and to a greater degree. After the dropped wrist has existed for some time, a swelling appears on the back of the hand, due partly to displacement of the bones of the wrist and partly to changes in the synovial membranes. There is seldom any definite disturbance of sensation. Another variety, which may occur independently of, or in association with, the forearm distribution, is that in which the loss of power affects the small muscles of the hand. The interossei and muscles of the thenar eminence waste, and the signs closely resemble those of progressive muscular atrophy. Only in severe cases does the paralysis extend beyond the forearms. In rare instances a spasm of the muscles occurs. In some cases there are symptoms of disturbance of the central nervous system, such as headache, vomiting, mental impairment, and optic neuritis; paralysis of cranial nerves has occasionally been observed. Recovery generally takes place very slowly, and the patient is very liable to relapse if he again comes under the influence of the poison.

POST-DIPHTHERITIC NEURITIS

Post-diphtheritic paralysis occurs in children and adults, but more often in the former. The first signs may show themselves a few days after the infection, or may not appear for several weeks; the usual period of onset is about a fortnight after infection.

Symptoms.—In the majority of cases paralysis of the soft palate is the first symptom, as evidenced by the regurgitation of fluids through the nose, and the presence of a “nasal” voice.

The ocular group of muscles is generally the next to be affected, and though any of these may

be paralysed, the external rectus and the ciliary muscles are those most frequently weakened. In severe cases the paralysis is more generalised, and there may be loss of power in all the limbs.

Of particular importance is the liability of the vagus and the phrenic nerve to the effects of the toxin.

The knee-jerks are generally lost early, and indeed they are often absent after an attack of diphtheria without any other symptoms of neuritis. There may be ataxy of the limbs, and sensory symptoms, such as pain, tingling, and anæsthesia, may be complained of. Vomiting is sometimes a troublesome symptom.

Course and prognosis.—Many cases run an uneventful course, and power gradually returns. When the paralysis does not spread beyond the soft palate and the ocular muscles, recovery often takes place quickly, but when the limbs are affected the duration is much longer, and it may be several months before power is restored. Signs that the power of the heart or of the diaphragm is impaired always put a grave aspect on the case. "Bulbar crises" characterised by failure of heart and respiration may occur, and are often associated with paralysis of the diaphragm (Pasteur).

Pathology.—The toxin appears to affect the whole of the lower motor neuron, and a parenchymatous degeneration of the myelin sheath of the nerves seems to be the most important lesion found after death (F. E. Batten).

Changes in the motor nuclei of the cord and bulb have, however, also been described, and alterations in the structure of the motor cells of the anterior horns of the cord have been noted in animals at quite an early stage of diphtheria intoxication.*

Fatty degeneration has been observed in the

* A. G. R. Foulerton and Campbell Thomson, *Edin. Med. Journ.*, Jan., 1902.

muscle tissue of the heart and diaphragm, and also in other viscera, and is regarded by Dudgeon as the essential cause of the acute cardiac failure which is apt to occur in these cases.

Diagnosis of peripheral neuritis.—Peripheral neuritis has to be distinguished from the various forms of *myelitis*. The main distinguishing features are the signs of a lower neuron lesion, combined with absence of sphincter trouble. A transverse myelitis above the lumbar enlargement is associated with the signs of an upper neuron lesion together with loss of control over the sphincters.

Other important differences are in the distribution of the anæsthesia and in the formation of bedsores.

In transverse myelitis there is anæsthesia below the level of the lesion, but in neuritis the sensory loss is confined to the limbs and does not extend to the trunk. The acute bedsores that are so commonly associated with myelitis do not occur in neuritis.

A myelitis of the lumbar enlargement may superficially resemble a peripheral neuritis, inasmuch as the knee-jerks may be lost, and the muscles of the thighs and legs may waste from destruction of the motor cells in the cord, but the complete loss of control over the sphincters which occurs in these circumstances is still a distinguishing feature.

From *acute anterior poliomyelitis* the chief distinctions are the alterations of sensation and the symmetry of the paralysis as compared with the irregularly scattered lesions of poliomyelitis. There are, however, some cases in which it is very difficult to arrive at a differentiation.

From *Landry's paralysis* there is also a difficulty, in some cases, in distinguishing peripheral neuritis, though in characteristic examples of Landry's disease the absence of any alteration of

sensation, of wasting, and of electrical changes in the muscles are distinguishing features.

The symptoms in a certain number of cases of neuritis closely simulate those of *locomotor ataxy*. The resemblance occurs when incoordination is a prominent feature, and where this exists in combination with absent knee-jerks and the presence of pains in the legs, tabes is apt to be diagnosed. Some cases of alcoholic neuritis closely resemble tabes in these particulars, as also do some cases of neuritis due to diabetes, in which the similarity may be marked by the presence of gangrenous ulcers on the feet, but the Argyll-Robertson pupil of tabes, and the loss of power with the altered electrical reactions in neuritis, generally assist the diagnosis.

From *hysterical paralysis* neuritis can be differentiated by the absence of knee-jerks and by the presence of wasting and changes in the electrical reactions of the muscles.

Prognosis.—The prognosis varies with the nature of the cause and the possibility of its removal. In general the chances of recovery of power are good if the cause can be removed, but the progress is often very slow.

In alcoholic neuritis, when the cause is removed, improvement generally takes place, except in the most severe cases, in which death may occur from general exhaustion or cardiac failure.

Signs of impairment of the functions of the vagus or phrenic nerves are always grave, and the special liability of these to be damaged in post-diphtheritic paralysis has been already mentioned.

Treatment.—It is obvious that, whenever possible, the cause must be removed at once.

Little can usually be done to shorten the process, but salicylate of soda and iodide of potassium sometimes appear to have favourable effects. After the acute symptoms have subsided, strychnine is probably the most useful drug. When

the pains are severe, local applications of heat and liniments containing chloroform and belladonna are useful, and occasionally alternate successive applications of heat and cold give relief. Phenacetin, antipyrin, antifebrin, aspirin, and salicylates are the drugs that are most useful as anodynes. In the worst cases recourse must be had to morphia.

Massage and electrical treatment should be commenced early, and care must be taken to prevent the paralysed limbs from assuming faulty positions, especially with regard to the feet.

In the post-diphtheritic cases it is most important to keep the patient at rest till the danger of cardiac failure is over, for fatal syncope may result from the patient merely sitting up in bed. During the attacks of respiratory failure which are apt to occur in the diphtheritic cases, artificial respiration may tide over the time of danger.

When lead has caused the neuritis, its elimination should be hastened by the regular administration of saline purgatives, of which magnesium sulphate is the one most often chosen. Iodide of potassium is also supposed to assist the elimination of both lead and arsenic, but the results of analyses of the excreta seem to show that it has not much influence, at any rate so far as the elimination of lead is concerned.

CHAPTER X

TUMOURS OF NERVES (NEUROMATA)

NERVE trunks are sometimes the seat of tumours, which are usually all grouped under the clinical title of neuromata, though their structure varies and is indeed seldom that of nerve tissue. Those most frequently met with are neuro-fibromata, which form fusiform swellings on the nerve trunks, and are very liable to undergo myxomatous degeneration. They may grow from the cranial or the spinal nerves, and are often multiple.

Plexiform neuromata (Fig. 22) are rare. They form more diffuse tumours than the neuro-fibromata and feel "like a bag containing a number of tortuous, irregular vermiform bodies, soft to the touch and mobile" (Bland-Sutton).

Closer examination shows that multiple connective-tissue tumours have formed along the course of the nerves. Of the cranial nerves the trigeminal is the most frequently attacked, but the growth may occur in any nerve, cranial or spinal.

Ganglionic neuromata are very rarely met with. They are formed of nerve cells, nerve fibres, and neuroglia.

Neuro-fibromata may arise in great numbers from the twigs of the cutaneous nerves and constitute the condition known as molluscum fibrosum, which, when associated with freckles or a more diffuse pigmentation of the skin, is known by the name of Recklinghausen's disease.



Fig. 22. — Plexiform neuromata affecting the roots of the chorda equina and anterior crural nerve. (*Bland-Sutton.*)

Symptoms.—The symptoms depend largely on where the tumours happen to be situated. Growths on sensory nerves may cause pain and various disturbances of sensation; among the most painful are those which arise on the nerve-endings of an amputation stump.

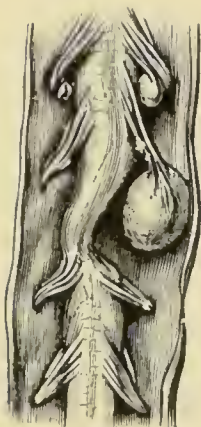


Fig. 23. — Cervical segment of the cord represented in Fig. 22. A nodule on one of the cervical roots compressed the cord and led to fatal paraplegia. (Bland-Sutton.)

Pressure on spinal roots may cause weakness in the corresponding muscles, which may be followed later by signs of paraplegia as the tumour invades the spinal cord (Fig. 23).

Prognosis.—Tumours growing from nerves are nearly always simple, and if removed show no tendency to recur.

There is, however, some danger of sarcomatous development in the case of molluscum fibrosum.

Death may be caused from pressure on the cord or brain.

Treatment.—When the tumour is accessible and causing unpleasant symptoms it should be removed, but when the growths are multiple the difficulties are naturally greater, and in the molluscum fibrosum variety nothing can be done.

SECTION III.—THE MYOPATHIES (MUSCULAR DYSTROPHIES)

CHAPTER XI

THE MYOPATHIES; PERONEAL TYPE OF ATROPHY

THE MYOPATHIES

UNDER the term “myopathies” is comprised an important group of cases in which the failure of vitality appears to arise in the muscles themselves. No definite changes have been found in the nervous system, and the essential feature of these cases is atrophy of the muscles, with which, however, there may be some accompanying enlargement.

Morbid anatomy.—Atrophy of some fibres, hypertrophy of others, and excess of fat and fibrous tissue are the principal changes observed in the affected muscles. While all these cases seem to be pathologically similar in their want of vitality, they vary considerably in their clinical appearances. Among the principal forms are—

1. The pseudo-hypertrophic form.
2. The facio-scapulo-humeral form, known as the Landouzy-Déjérine type.
3. The juvenile form of Erb.

As might be expected from their common pathological basis, no sharp line can be drawn between these different forms. Cases in which atrophy is the dominant feature often show traces of pseudo-hypertrophy, while cases of pseudo-hypertrophy invariably show some signs of atrophy.

Like the parallel cases of spinal origin, the

myopathies are very liable to occur in several members of a family.

I.—PSEUDO-HYPERTROPHIC PARALYSIS

Etiology.—This disease begins usually between the ages of four and ten. It exhibits a distinct tendency to run in families, and is usually transmitted by females and inherited by males.



Fig. 24.—Case of pseudo-hypertrophic paralysis. (*Pierre Marie*.) Observe the enlargement of the muscles of the thighs and calves as compared with the thin arms.

Symptoms.—Clumsiness, frequent falls, and a difficulty in going upstairs are generally among the early symptoms, all of which seem the more strange to the parents since the limbs, especially the calves, often appear to them to be unusually well developed. Examination, however, shows that



PLATE IV.—Instantaneous Photograph showing “High-steppage”
Gait in Pseudo-hypertrophic Paralysis.

(From a Case under the care of Dr. George Ogilvie.)

the legs are weak in spite of their increased size, the latter proving to be a pseudo-hypertrophy due to overgrowth of fat and connective tissue which have displaced the wasted muscle fibres.

Distribution of hypertrophy.—The muscles in which some enlargement can most usually be seen are the muscles of the calf, the quadriceps extensor, the glutei, the triceps, and the infraspinati. They are hard and firm to the touch, but despite this they are weaker than they should be. In some cases part of a muscle is enlarged and part atrophied.

Muscles most commonly atrophied.—Some muscles are from the first atrophied and show no sign of enlargement; among them the most constant are the lower half of the pectoralis major and the latissimus dorsi. Others in which atrophy can very frequently be detected are the biceps, the flexors of the knee, and the peronei.

Certain muscles generally escape, e.g., the muscles of the face, forearms, and hands, but occasionally some enlargement of the tongue may be observed.

Attitude.—The failure of muscular power makes it difficult for the patient to stand, walk, or rise from the ground, and, in endeavouring to perform these actions, fairly characteristic attitudes are assumed. In standing the feet are kept wide apart in order to obtain a broader base, while the trunk is thrown backwards (lordosis) in order to keep the line of gravity from falling too far forward. The arms are generally flexed and adducted.

In walking the same position is preserved, and the patient waddles from side to side, with the feet turned out, and with a "high steppage" gait (Plate IV.).

In rising from the supine position the patient first rolls over, then takes his fixed points from his hands and toes, and finally helps himself up by placing his hands on his thighs.

All these positions are due to attempts to com-

pensate for the weakness of the affected muscles, and therefore they vary widely in degree in different cases.

Reflexes.—Wasting of muscles leads to the loss of any reflexes which they may normally serve;



Fig. 25.—Deformities arising in the course of pseudo-hypertrophic paralysis. (*Pierre Marie*.) This is the brother of the patient in Fig. 24.

thus the knee-jerk diminishes as changes occur in the quadriceps extensor muscle.

Deformities.—Talipes of the feet and curvature of the spine are both often met with (Fig. 24).

II.—FACIO-SCAPULO-HUMERAL TYPE

In the facio-scapulo-humeral type (known also as the Landouzy-Déjérine form) the atrophy, like that of pseudo-hypertrophic paralysis, generally begins in childhood. It is often spoken of as the

"infantile form" of myopathy in contradistinction to the "juvenile form" which begins in later life.

The name "facio-scapulo-humeral" shows the chief distribution of the wasting.

In the face the orbiculares palpebrarum and the orbicularis oris are commonly weakened, and

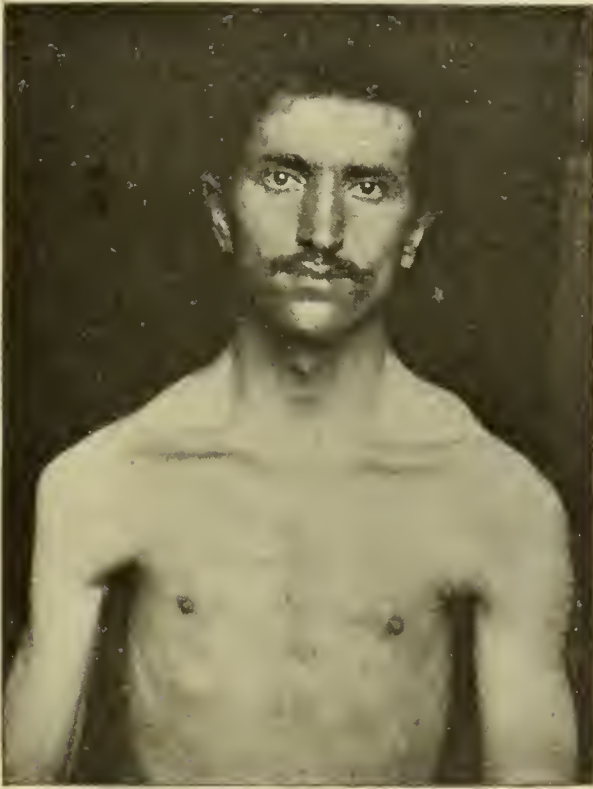


Fig. 26.—Appearance of neck produced by displacement of the scapulae and dropping of the shoulders in a case of myopathy. (*Pierre Marie.*)

as a result of the weakness of the latter muscle, together with others at the corner of the mouth, quite a characteristic expression is given to the features.

Next, the muscles of the shoulder-girdle and upper arm atrophy, and here the trapezius (both upper and lower divisions), latissimus dorsi, serratus magnus, pectorals, triceps, and biceps generally suffer early. The wasting of the serratus

trapezius
lat. dorsi
serratus magnus
pectorals
triceps
biceps

magnus causes the vertebral border of the scapula to tilt whenever the arm is lifted.

The forearms generally escape, the hands always.

From the shoulder-girdle the wasting spreads to other muscles of the trunk and limbs. The



Fig. 27.—Case of myopathy showing enlargement of the infraspinati. (*Pierre Marie.*)

muscles of the back and hip become weak, causing lordosis and a waddling gait like that in the pseudo-hypertrophic form, while wasting of the other muscles is accompanied by difficulty in walking and deformities of the feet. Some muscles or parts of muscles are often found to be enlarged, and thus a relationship is established between this and the pseudo-hypertrophic form.

III.—ERB'S JUVENILE FORM

In the juvenile form the wasting is generally noticed rather later in life, but nearly always before the boy is out of his teens. The wasting generally begins about the shoulders and then



Fig. 28.—Case of myopathy showing wasting of the muscles of the trunk causing the deformity of the trunk and the waist known as the "*taille de guêpe*." (*Pierre Marie*.)

spreads to the trunk and legs; the muscles of the face are also affected sometimes. Enlargement of some muscles may be observed.

Diagnosis of the myopathies.—From progressive muscular atrophy of spinal origin the chief points of distinction are:

- (1) The age at which the disease begins.
- (2) The distribution of the wasting. In the

myopathies now under consideration the muscles of the trunk waste early, the hands always escape, and the deltoid is often normal; whereas in progressive muscular atrophy of spinal origin the hands and the deltoid are among the earliest to exhibit signs of the disease.



Fig. 29.—Curvature of the spine occurring in the course of myopathy. (*Pierre Marie.*)

(3) The myopathies tend to occur in several members of a family, and when this peculiarity is present the spinal variety may be excluded.

(4) Hypertrophy either true or false is not found in the cases of spinal origin.

(5) Fibrillary tremors are common in the spinal cases and rare in the myopathies.

The distribution of the wasting, absence of sensory changes, and the character of the electrical



PLATE V.—Case of Myopathy showing the
Characteristic Attitude of Lordosis.

reactions serve to differentiate these cases from peripheral neuritis.

The electrical changes shown in the myopathies are usually those of diminished reactions to both the faradic and galvanic currents. There is usually no definite "reaction of degeneration."

Prognosis.—All these different forms of the myopathies tend to progress slowly, but here and there arrest takes place or the progression is so slow that it can be ignored.

The danger to life lies in the atrophy extending to the muscles of respiration, and consequently in the failure to combat successfully any pulmonary complications, such as bronchitis, pneumonia, and tubercle.

Treatment.—Every effort to arrest the wasting must be made. Massage, moderate exercise, and electricity should all be used to keep up the nutrition of the muscles, and must be persevered with if any success is to be obtained.

THE PERONEAL TYPE OF MUSCULAR ATROPHY

The early descriptions of this variety, which is characterised by wasting of the muscles of the legs and forearms, are especially associated with the names of Charcot, Marie, and Tooth.

Etiology.—Nothing definite is known of the causation. The symptoms generally first appear in children or young adults. There is a tendency for several members of a family to be attacked, and cases of heredity have occasionally been recorded.

Pathology.—The exact pathology is still doubtful, and in the light of present knowledge it is convenient to look upon this variety as an intermediary form between the atrophies of muscular and spinal origin. Sclerosis of the posterior and, to some extent, of the lateral columns of the cord has been observed, and the occurrence of a

curious hypertrophic form of neuritis has been occasionally noticed.

Symptoms.—The onset is very gradual. At first the peronei and extensors of the toes become weak, and later on the calf muscles waste, so that the size of the leg contrasts markedly with that of the thigh, which generally remains normal for some time (Fig. 31). Clubbing of the feet occurs and is frequently an early sign.

When the thigh muscles remain normal the knee-jerks may be retained. After a time, wast-



Fig. 30.—Section of spinal cord showing degeneration of the posterior column, from a case of peroneal atrophy. (*Pierre Marie.*)

ing takes place in the small muscles of the hand and spreads to the muscles of the forearm. Deformities resulting in the "claw" hand may eventually arise. In some cases the disease does not make any further progress, but in others the wasting gradually spreads to the thigh and trunk.

Disturbances of sensation are uncommon, but pain and anæsthesia of the limbs have occasionally been observed.

Diagnosis.—The main points upon which reliance has to be placed are slow symmetrical wasting of the legs, accompanied by clubbing of

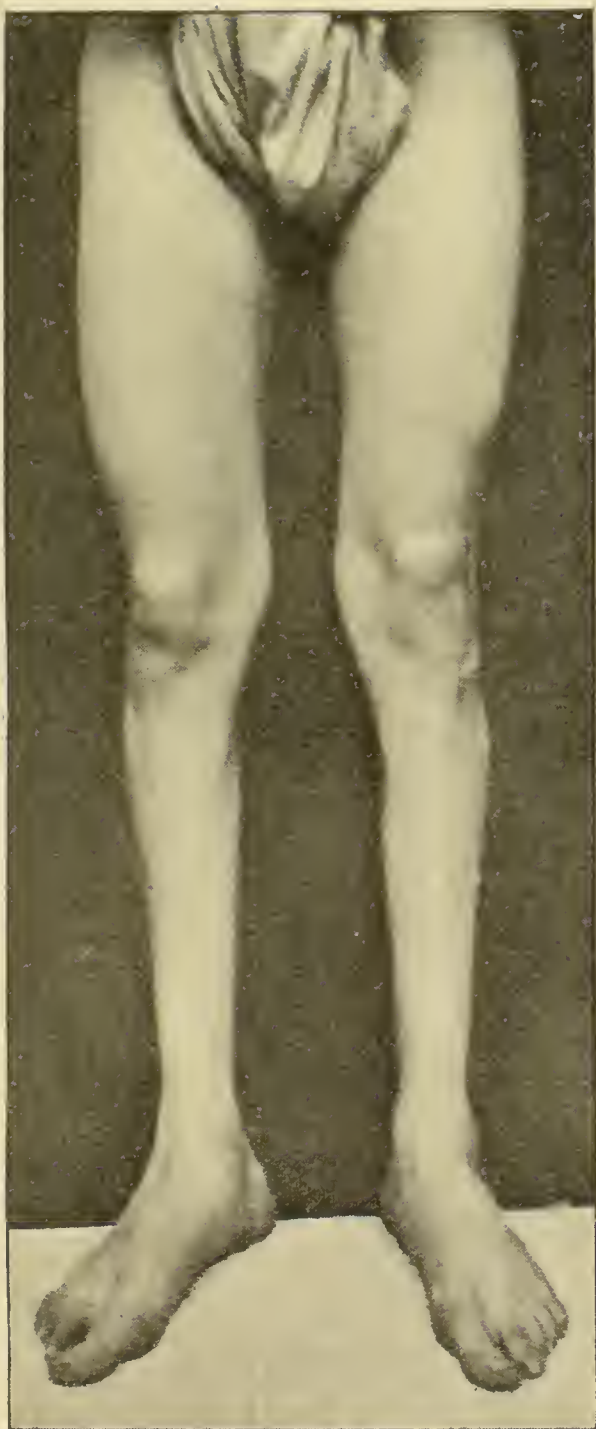


Fig. 31. — Case of peroneal atrophy. Note the wasting of the muscles below the knee.

the feet, followed later by wasting and deformities in the hands and forearms, occurring in children and young adults, though from what has already been said it will be seen that cases may occur in which there is doubt about the underlying spinal and neural changes.

Prognosis.—The prognosis as regards recovery is bad, but as regards life it is more favourable as compared with the other forms of myopathy, for in the peroneal type the atrophy often ceases to extend.

Treatment.—Massage, electricity, tonics, and attention to the general health are the only methods of treatment applicable to this disease.

SECTION IV.—DISEASES OF THE SPINAL CORD

CHAPTER XII

CLASSIFICATION

IN practice the patient comes with certain complaints which have to be investigated and referred to their proper causes. But before this can be done it is necessary that some knowledge should be acquired of the principal lesions which are likely to be met with, and for the purpose of gaining this knowledge classifications are utilised.

It is true that such classifications are necessarily often artificial and not altogether scientific, but they nevertheless form a useful working basis and serve as an aid to memory, while they can do but little harm so long as their provisional nature is duly emphasised.

One such method of dealing with diseases of the spinal cord is to classify them according to the anatomical distribution of their lesions, and taken in this way the disorders at once separate themselves into two large groups, viz.:

1. Those in which the morbid changes are confined within the limits of certain columns, *i.e.*, the so-called **system lesions**; and

2. Those in which the morbid changes are distributed irregularly through the substance of the cord quite irrespective of column formation, *i.e.*, **non-system lesions**.

THE SYSTEM LESIONS

Reference to the accompanying diagrams shows the chief systems, and these may be attacked

singly or in numbers, and accordingly there may be single or combined system lesions. Of the single

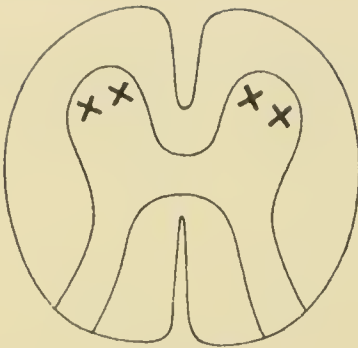


Fig. 32.

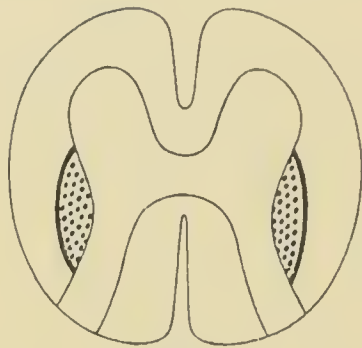


Fig. 33.



Fig. 34.

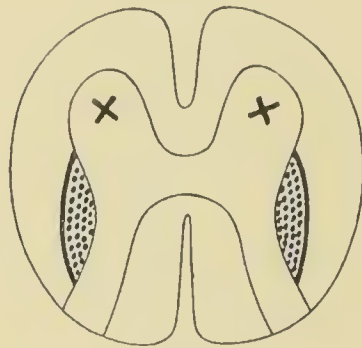


Fig. 35.



Fig. 36.

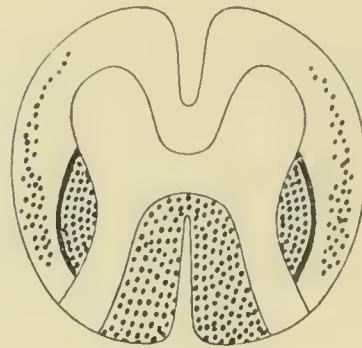


Fig. 37.

system lesions, where only one column is attacked, we may recognise the following:

1. Disease of the cells of the anterior horns, as exemplified in infantile paralysis and progressive muscular atrophy (Fig. 32).

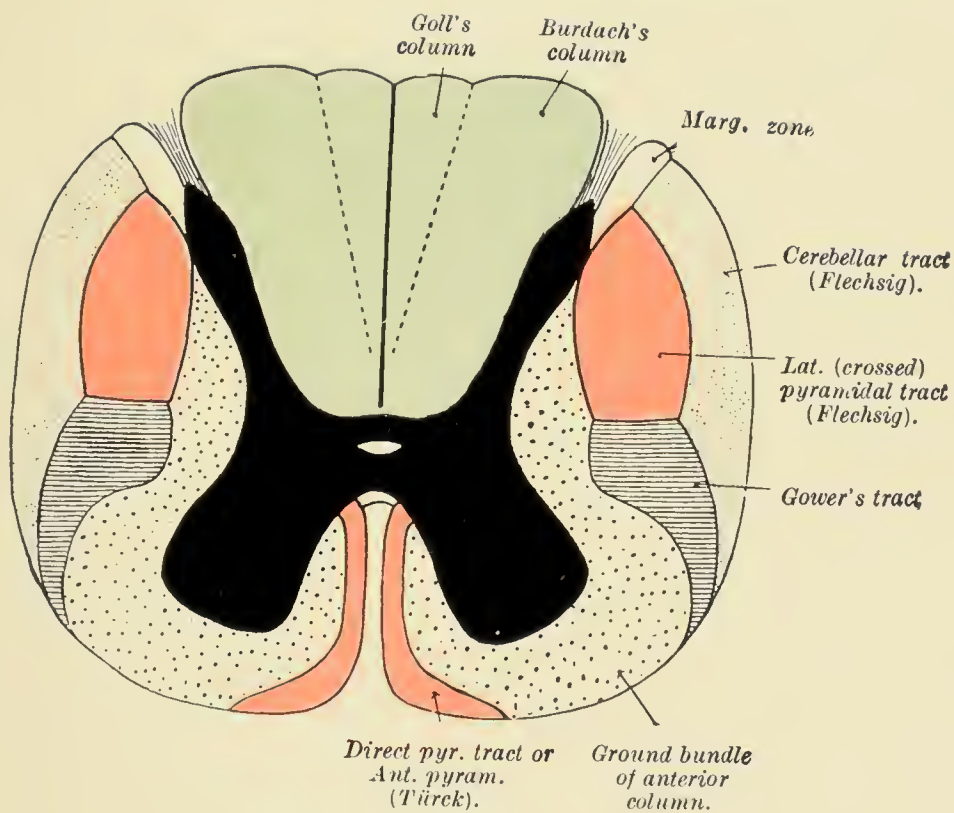


PLATE VI.—Tracts of the Spinal Cord. (Morat.)

2. Disease of the lateral (crossed pyramidal) tracts, as exemplified in lateral sclerosis (Fig. 33).

3. Disease of the posterior columns, as exemplified in locomotor ataxy (Fig. 34).

Combinations of these may occur as follows:—

(a) Disease of the lateral columns together with those of the cells of the anterior horns, as exemplified in amyotrophic lateral sclerosis (Fig. 35).

(b) Diseases of the lateral columns together with those of the posterior columns, as exemplified in ataxic paraplegia (Fig. 36).

(c) Diseases of the lateral columns together with the posterior columns, cerebellar tracts, and (sometimes) antero-lateral tracts, as exemplified in Friedreich's disease and subacute combined degeneration (Fig. 37).

Thus the groundwork of the combined lesions making up amyotrophic lateral sclerosis and ataxic paraplegia can be deduced without any special effort of memory by combining the symptoms of lateral sclerosis of the lower limbs with progressive muscular atrophy of the upper limbs in the case of the first, and with ataxia in the case of the second.

IRREGULAR AND NON-SYSTEM LESIONS

In these no special predilection is shown for the various columns, and the classification has to be based on the pathological possibilities. The main ones are:

Inflammations, e.g., myelitis—transverse or disseminated.

Vascular lesions, e.g., thrombosis and hæmorrhage.

Diffuse and disseminated degenerative processes, e.g., syringo-myelia and disseminated sclerosis.

Syphilitic lesions.

Tumours.

Pressure from without, as in spinal caries,
meningeal disease, injuries, etc.

This method of classification is, of course, very far from perfect. For instance, the degeneration in tabes dorsalis is not entirely confined to the posterior columns. Nevertheless the classification is one that proves useful to students, since it serves as a model which they can supplement or vary at their discretion, and at the same time it helps to a considerable extent to demonstrate the relationship which exists between the different diseases.

CHAPTER XIII

PARAPLEGIA AND BULBAR PARALYSES

PARAPLEGIA

PARAPLEGIA is the clinical term usually applied to a condition of paralysis of both legs, though it is equally applicable when the arms are also involved, as, for instance, in a case of fracture of the spine in the cervical region.

The presence of paraplegia indicates that the transmission of impulses along the motor fibres has been interrupted, and its causes are best studied by making a diagram of the motor paths and then considering the whereabouts and nature of the possible interruptions.

1. Lesions of the peripheral nerves.—Peripheral neuritis from alcohol, arsenic, or any other cause may give rise to complete loss of power in both legs, and the position of the lesion has to be decided by the characteristics of the lower neuron paralysis, and then differentiated from diseases of the anterior cornua.

2. Lesions of the spinal cord.—Disease of the cells of the anterior horns in the lumbar region will, if symmetrical, cause paraplegia.

Here the paralytic symptoms, being of the lower motor neuron type, are similar to those of peripheral neuritis, but there will be no sensory changes such as occur when the peripheral nerves are affected. As a matter of fact, a paraplegia of symmetrical distribution due to a lesion limited to the cells of the anterior horns

is rare. In poliomyelitis a symmetrical lesion is most unusual, while in progressive muscular atrophy degeneration of the motor cells of the lumbar region is seldom a predominant feature.

Any lesion of the spinal cord which implicates the *lateral columns* will cause some degree of paraplegia.

These may be divided into:

(a) Those which occur primarily in the substance of the cord.

(b) Those which press on the cord from without.

(a) Those of the *substance of the cord* may be transverse lesions (complete or incomplete), such as arise from myelitis, hæmorrhages, thrombosis, tumours, and syringo-myelia, or they may be "system lesions" in which definite columns are attacked, such as in primary lateral sclerosis and ataxic paraplegia.

(b) *Pressure from without* may be due to fracture-dislocation of the spine, caries, and tumours.

In all these cord lesions, except when the lumbar enlargement is diseased, the paralysis of the legs is that of the upper neuron type, as shown by the increased reflexes, rigidity, ankle clonus, and extensor response of the plantar reflex, while there is no wasting (except from disuse) and no change in the electrical reactions of the muscles. The action of the bladder varies; when the lesion extends across the cord there is usually trouble, but in some of the system lesions little or nothing may be noticed.

3. Lesions of the brain.—(a) *Sub-cortical*.—As we pass above the cord the motor fibres diverge to the cortex of the brain, and therefore between the region of the pons and the cortex it is unlikely that any single lesion would injure them on both sides (Plate I.).

Symmetrical lesions are naturally uncommon,

hence hemiplegia rather than paraplegia is the usual result of disease in this area; but it does occasionally happen that two vascular lesions succeed one another and cause a "double hemiplegia," which is to all intents and purposes a paraplegia.

(b) Cortical.—In the cortex the motor areas for the legs are not far from each other, and any injury to the vertex of the brain is liable to damage both the leg centres and cause paraplegia. Some of the paraplegias of infancy arise in this way through injuries at birth (*see* Cerebral Diplegia), but it is very seldom that paraplegia arises in this manner in later life.

(c) Hysteria.—Paraplegia of a most complete type may be of hysterical origin, and its cause is then probably situated in the cerebral cortex.

Some of these cases give rise to great difficulty in diagnosis, for though they can usually be easily distinguished from lower neuron lesions, such as neuritis, where the wasting and the loss of reflexes make the condition clear, they are often confused with cord lesions, and especially with those due to commencing disseminated sclerosis, in which the symptoms are apt to vary in the same extraordinary fashion as they do in functional disease.

It is in these doubtful cases that the extensor response of the plantar reflex is so valuable, for increased knee-jerks, with perhaps slight ankle clonus, may be present in both.

Further particulars of differential diagnosis of disseminated sclerosis will be found in Chapter XX.

For the **prognosis** of paraplegia, which, of course, depends upon its cause, reference may be made to the chapters dealing with the different diseases in which paraplegia occurs.

SENILE PARAPLEGIA

In elderly people some weakness of the legs, accompanied, perhaps, by some difficulty in exercising full control over the sphincters, may occur

from anæmia of the cord due to atheroma of the spinal arteries.

INTERMITTENT CLAUDICATION

Another difficulty in walking—due, it is supposed, also to atheroma—is that known under the name of *intermittent claudication*, or *limping*, which is characterised by sudden attacks of lameness, often accompanied by cramps, blueness of the extremities, and sensations of tingling. After walking a variable distance the patients are pulled up suddenly with cramp, which usually passes off again with rest. The symptoms in these cases are often attributed to impaired nutrition of the muscles consequent on the arterial disease, and absence of arterial pulsation and the presence of gangrene in the feet may be associated conditions.

Some authors, however, think that the symptoms depend on changes in the cord or the brain.

Sir William Gowers* mentions instances in which intermitting lameness and intermitting difficulty of reading (dyslexia) are associated—a combination of serious import, since in most cases it indicates the presence of a widespread arterial degeneration.

BULBAR PARALYSES

Paralyses of bulbar origin, in which the tongue, pharynx, and larynx are affected, may, like other paralyses, be of upper or lower neuron type.

1. *Upper neuron type*.—In this form the motor fibres are interrupted in their course from the cortex to the medulla, but, in order to produce any permanent effect on the bulbar functions, the interruption must be bilateral. A unilateral lesion, *e.g.*, a hæmorrhage into the internal capsule, though it may affect the movements of one half of the tongue and palate, will not as a rule cause any permanent loss of articulation and difficulty

* "Manual of Diseases of the Nervous System," I., 341.

in swallowing. On the other hand, a bilateral lesion will, by cutting off the motor fibres from both sides of the cortex, deprive the nuclei of voluntary innervation, and in such a case the movements of the tongue, palate, and larynx may be greatly impaired.

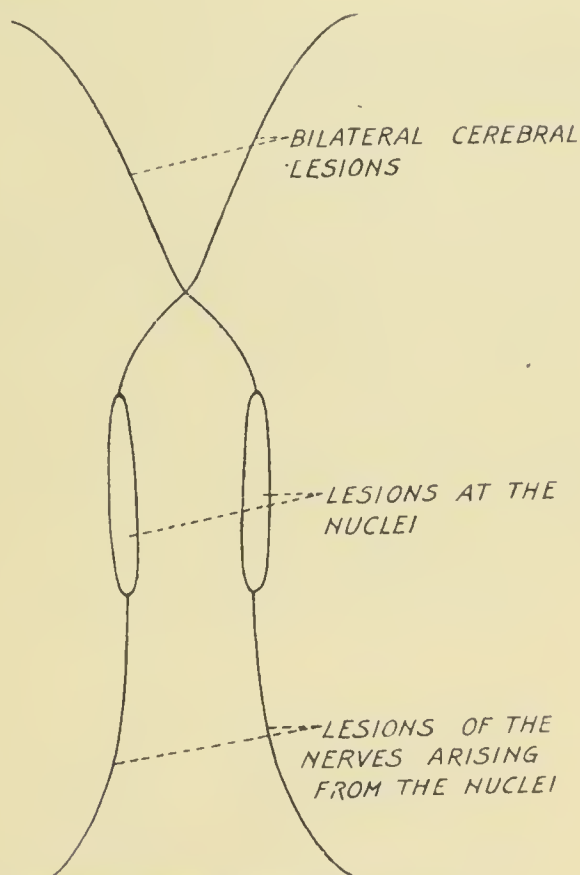


Fig. 38.—Diagram showing different situations in which bulbar paralysis may be produced.

Bilateral lesions of cortical origin may be seen in severe cases of cerebral diplegia in children: they also occur in amyotrophic lateral sclerosis and in cases where a hæmorrhage in the region of the internal capsule is followed by another in a similar position on the opposite side. The bulbar symptoms due to upper neuron lesions are often termed pseudo-bulbar paralysis, to distinguish

them from the lower neuron lesions in which the functions of the nuclei themselves are lost.

The same rules for upper motor neuron lesions apply here as elsewhere. The power of voluntary movement is diminished. There is inability to pout the lips in order to blow or whistle, the tongue cannot be protruded or thrust into the cheeks, and articulation is very indistinct. Mastication and swallowing become difficult, the saliva dribbles out between the paralysed lips, and, in swallowing, fluid may regurgitate through the nose. The reflexes of the palate, pharynx, and larynx are preserved, a jaw clonus may be present, and there are no wasting and no changes in the electrical reactions of the paralysed muscles.

2. *Lower neuron lesions.*—Here the disease may be in the nuclei or in the nerves arising from them. The nuclei of the bulb, from which the tenth, eleventh, and twelfth nerves arise, are subject to the same diseases as nuclei elsewhere. They may be grouped as follows:

(1) *Inflammations.*—Acute inflammatory affections of the bulbar nuclei are very rare, but a condition analogous to that which takes place in the cord during anterior poliomyelitis (infantile paralysis) has been known to occur.

(2) *Degenerations.*—Chronic degeneration of the bulbar nuclei constitutes the disease known as progressive bulbar paralysis. It is in reality a part of progressive muscular atrophy and is described under the heading of that disease (Chapter XV.).

(3) *Vascular lesions.*—(a) Hæmorrhage, (b) thrombosis, (c) embolism. Hæmorrhage of any size into the bulb naturally soon causes death, but thrombosis or embolism, more especially thrombosis of some small branches, may cause symptoms of disease of the bulbar nuclei. The more sudden onset of the symptoms serves to distinguish such cases from

the progressive bulbar variety; their course depends upon the temporary or permanent effects which the lesion produces.

(4) Pressure.—Tumours in the neighbourhood of the medulla may give rise to bulbar symptoms from pressure.

When the bulbar paralysis is due to injury of the nerves after they have left the nuclei the symptoms are generally unilateral, and, moreover, the spinal portion of the spinal accessory will in all probability be also injured, in consequence of which the additional symptoms of paralysis of the sterno-mastoid and trapezius will be present.

Examples of neuritis in which the symptoms are symmetrical and of bulbar distribution are met with in cases of post-diphtheritic paralysis. Another disease in which bulbar signs are also often prominent is myasthenia gravis, the pathology of which is at present not certainly known, though there is some evidence to show that the morbid changes may possibly commence in the muscles themselves (*see* Chapter XXXVI.).

CHAPTER XIV

ACUTE POLIOMYELITIS (INFANTILE PARALYSIS)

ACUTE poliomyelitis, known clinically also as infantile paralysis, is the result of acute inflammatory changes taking place in the motor cells of the anterior horns of the spinal cord.

Etiology.—Children between the years of three and eight are the chief sufferers, but the disease is by no means unknown amongst adults. That the disease is due to an acute infection is shown by the occasional incidence of epidemics and by the occurrence of the greater number of cases in the summer months. Various micro-organisms have been isolated, but at present no particular one can with certainty be identified with the disease, and it seems more than probable that their nature varies in different cases.

That an acute infection can give rise to infantile paralysis in one child and to something entirely different in other children has been shown by William Pasteur,* who observed a family the different members of which, apparently, had all been exposed to the same poison. Of the seven children comprising this family two were the subjects of infantile paralysis, one had thrombosis of cerebral vessels followed by hemiplegia, while the other four escaped with a general febrile attack accompanied by nervous symptoms of a transient character.

* *Trans. of the Clin. Soc. of London*, Vol. XXX.

Pathology.—Acute hyperæmia of the vessels, cell proliferation, and disintegration of the large motor cells are the chief conditions found in the early stages of the disease.

Thrombosis may be present, and has been thought by some to be the primary lesion. The whole subject has recently been reviewed by Farquhar Buzzard,* who concludes that the evidence at present available points to the essential lesion being an inflammation of the interstitial tissue of the central nervous system, the effects of which are visited mainly upon the grey matter on account of its greater vascularity and slighter resisting powers. Changes can, however, be recognised in the white matter in the shape of swollen axis cylinders and myelin sheaths, and clinical evidence of temporary disturbance of the function of the pyramidal tracts can sometimes be established. Signs of inflammation have been observed in the meninges also.

In the later stages, after the vascular disturbance has subsided, disappearance of motor cells and substitution of connective tissue are the chief features.

Symptoms.—The early symptoms are those of a general toxæmia; the child becomes fretful, restless, and is obviously unwell; the temperature is raised, and there may be a convulsion. The child seems to be in pain and dislikes being moved. In the course of a day or two it is noticed that one or more of the limbs are not being freely used. This gives rise to alarm and leads to an examination of the child.

The most striking feature at first is the presence of a flaccid paralysis of acute onset and irregular distribution. The nutrition of some of the motor cells in the anterior horns is suddenly and severely disturbed, and the corresponding

* Goulstonian Lectures on "Acute Infective or Toxic Conditions of the Nervous System," 1907.

muscles to which they contribute fibres consequently rapidly manifest all the signs of a lower neuron lesion. Groups of muscles are picked out irregularly in different limbs, but the legs suffer more frequently than the arms. Sometimes, but not often, some of the muscles supplied by the cranial nerves are paralysed, and cases in which power in the diaphragm has been lost have also been observed.

A little later, rapid wasting of the affected muscles becomes apparent, and their electrical reactions, if tested, will be those of degeneration. No disturbance of sensation will be found, and there is no loss of control over the sphincters.

Diagnosis.—The loss of power indicates a lesion of the motor tract, and the wasting and the grouping of the paralysis soon show that the lower neuron is at fault, *i.e.*, that the lesion is situated somewhere between the cells of the anterior horns and the muscles.

As acute diseases of the muscles giving rise to such symptoms are unknown, the issue is further narrowed to diseases of the peripheral nerves and of the anterior horns.

Peripheral neuritis can generally be excluded without much difficulty by the absence of pain and of sensory disturbance, and by the irregular distribution of the paralysis and its failure to correspond to the muscle areas of peripheral nerves.

Difficulty, however, occasionally arises in cases of multiple neuritis of acute onset in which no definite changes in sensation can, at any rate for a time, be demonstrated, but here again the symmetry is of great assistance.

Lesions of the muscles and peripheral nerves being excluded, the cells of the anterior horns remain to be considered. Here the only conditions that are likely to occasion confusion are those in which the function of the cells is very rapidly modified by toxins which give rise to cases coming

into the clinical group of *Landry's paralysis*. Here the patient rapidly loses power, as in infantile paralysis, but there is no definite wasting of muscles nor are there changes in their electrical

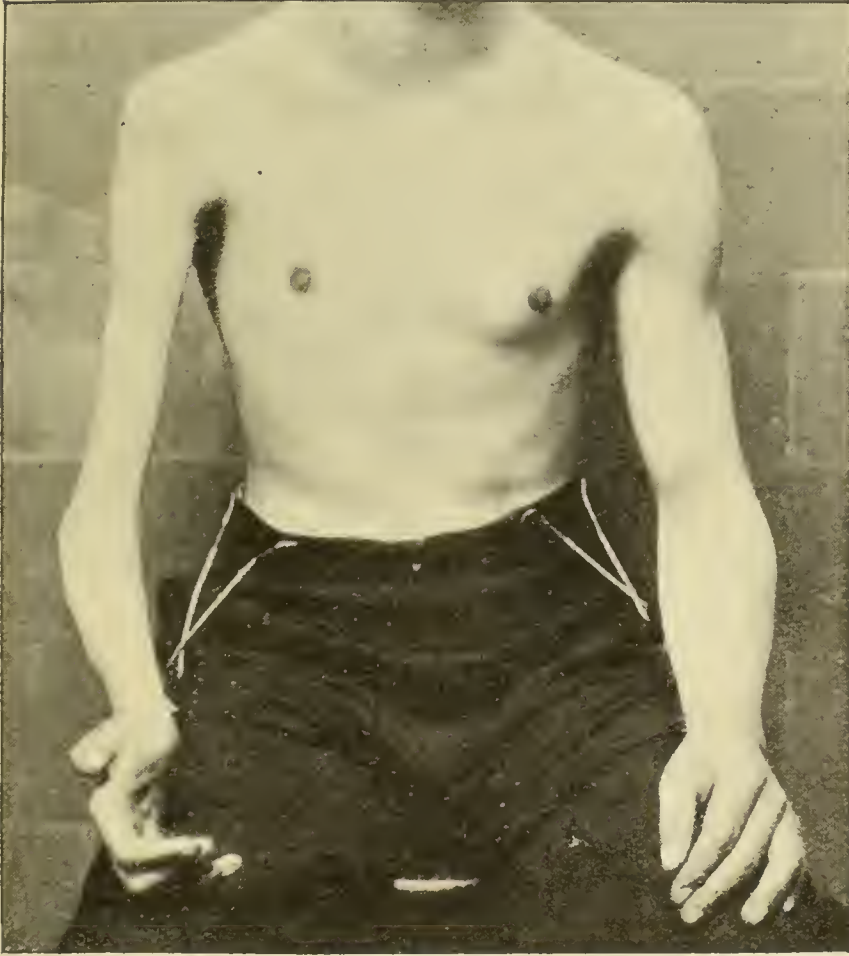


Fig. 39.—Wasting and deformity of the right hand and arm as the result of acute poliomyelitis in childhood.

reactions. Moreover, the loss of power in Landry's paralysis advances evenly upwards from the feet, while that of acute poliomyelitis is nearly always irregularly distributed.

Prognosis.—The diagnosis having been made, the physician must be prepared to indicate the course which the disease is likely to take. The

guide to the prognosis in these cases is to be found in the consideration of their morbid anatomy. If a group of cells from an affected area be examined, it will be seen that while some are entirely destroyed, others are badly injured, and others, again, on the borders of the diseased area have escaped. Those which are destroyed can never recover their function, but those in which the injury is less complete are capable of some degree of regeneration, and it is upon these latter that the ultimate degree of recovery depends.

As a general rule it may be said that, if all goes well, some considerable degree of improvement will take place, but as to how much, time alone can show. When the paralysis is extensive the growth of all the structures will be impaired. The limb remains stunted, wasted, and colder than its fellow, and, as time goes on, the muscular tissues contract and produce various kinds of talipes in the leg or corresponding deformities in the arm (Fig. 39).

Sometimes troublesome ulcers arise in the skin of the ill-nourished limb.

Treatment.—During the febrile stage the child should be kept in bed. The bowels should be opened by a dose of grey powder or calomel, repeated if necessary, and an anti-febrile mixture containing diaphoretics and diuretics may be given. At this stage little more can be done, but, as soon as the acute symptoms have subsided, efforts must be made to maintain the nutrition of the muscles until the damaged cells recover sufficiently to take on their functions. Massage and electricity are the most reliable methods to adopt, and nowhere is perseverance in treatment more necessary than in this disease. The treatment must be kept up for a very long time, with the object of improving those fibres which, though badly nourished, are still, by persistent stimulation, capable of being strengthened sufficiently to

aid in moving the limb. During this period every effort must be made to prevent contractions and deformities, by means of massage, passive movements, and the judicious use of splints. When they do arise, as unfortunately often happens in spite of treatment, the possibility of making a more useful limb by means of surgical methods may be considered.*

CHRONIC ANTERIOR POLIOMYELITIS

A chronic form of anterior poliomyelitis, differing from the chronic degeneration of progressive muscular atrophy by its non-progressive nature, is sometimes apparently met with clinically, *e.g.*, in some cases of chronic lead poisoning where distribution of the weakness and wasting corresponds to disease of anterior horns and roots, and not to that of peripheral nerves.

It is important to recognise the occasional occurrence of such cases, in order to avoid the error of diagnosing them as progressive muscular atrophy, and thereby giving a bad prognosis.

* An investigation, the results of which may have an important bearing on the causation of acute poliomyelitis, has recently been made at the Middlesex Hospital by Dr. W. Pasteur and Mr. A. G. R. Foulerton. A diplococcus was identified in the patient's cerebro-spinal fluid, and a subdural injection of the latter into two rabbits caused an ascending paralysis to develop in both on the forty-ninth and fiftieth days respectively after the injection, the diplococcus being again identified in the cerebro-spinal fluid of both animals.

CHAPTER XV

PROGRESSIVE MUSCULAR ATROPHY AND PROGRESSIVE BULBAR PARALYSIS

PROGRESSIVE muscular atrophy is the title given to those cases in which wasting of muscles results from a slow and progressive degeneration of the motor cells in the anterior horns of the spinal cord, or of their analogues in the medulla and mid-brain.

In order, however, adequately to understand the nature of the disease, it is necessary to take a rather wider view of its pathology and to regard it as an affection of the whole motor system (*i.e.*, of the cell bodies of both upper and lower neurons), in which the lower neurons are generally the more severely attacked.

Etiology.—The cause of this disturbance of nutrition is not known. Most of the cases occur towards middle life; more men suffer than women, and heavy manual labour sometimes appears to be connected with the onset of the disease. Syphilis is not generally regarded as having any special influence on the degeneration. All that can be stated is that some poison, the nature of which is at present unknown, causes a progressive degeneration to take place in the cell bodies of the motor neurons.

Symptoms.—The usual clinical picture of progressive muscular atrophy is due to the predominating degeneration of the cells of the lower motor neurons, those of the upper neurons being

usually affected to a less degree. The degeneration of these lower neurons does not always begin at the same level. Most often the cells of the cervical region of the cord are the first affected, and next in frequency those of the medulla. Less often the cells of the lumbar region are attacked first, and least often of all the first signs of the disease arise from degeneration of the nuclei of the ocular nerves.

When the disease begins, as it generally does, in the cervical region, the cells at either the upper or the lower end of the cervical enlargement generally degenerate first, and hence the wasting is first noticed in the muscles of the shoulder or of the hand. In the hand there is wasting of the interossei and of the muscles of the thenar and hypothenar eminences (Fig. 41). Depressions appear between the metacarpals, and the outline of the first of these bones becomes unduly prominent as the fibres of the first dorsal interosseous muscle (*i.e.*, the abductor indicis) shrivel.

The fingers can no longer be fully separated, and the power to abduct and oppose the thumb is lost. At the same time the hand gradually assumes a claw-like shape (*main en griffe*), and the thumb tends to rotate so that it looks like a fifth finger, and thus resembles the thumb of an ape. The reaction of the wasted muscles to faradism and galvanism is diminished and later may altogether disappear, but there are

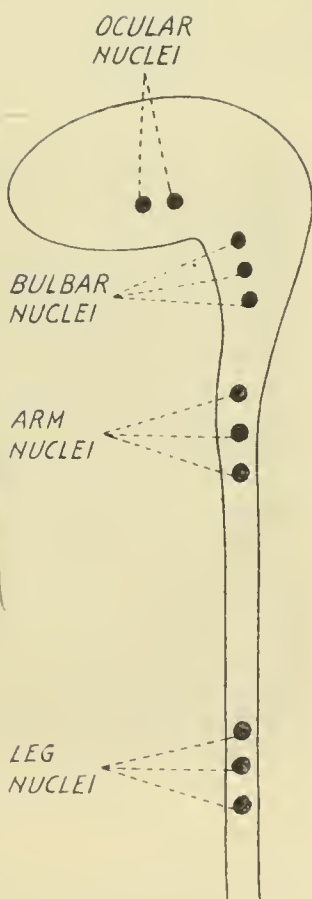


Fig. 40.—Diagram showing different positions in which progressive muscular atrophy may commence.

generally some fibres left which still show some reaction to faradism right up to the end, and hence the "reaction of degeneration" is not usually present.

The reflexes in the diseased areas disappear, because their arcs are broken by the degeneration of the motor cells.

As the motor cells are alone affected, there is



Fig. 41.—Wasting of hands in progressive muscular atrophy.

no loss of sensation, nor is there usually any pain beyond that of aching and stiffness, which may well be due to the changes taking place in the muscles. Fibrillary tremors are very often present.

After a time, perhaps a few months, wasting begins in the opposite hand, and as degeneration extends to other parts of the cord the muscles of the shoulder and trunk waste, and the weakness of the trunk muscles may imperil the adequacy of the respiratory movements, thus disposing to pulmonary complications.

In other cases the disease spreads upwards to

the nuclei of the bulb, thus giving rise to super-added symptoms of **progressive bulbar paralysis**, which is practically a progressive muscular atrophy in which the nuclei of the bulb are attacked.

In the bulb the cells chiefly affected are those of the tenth, eleventh, and twelfth nuclei; hence there is loss of power in the lips, tongue, pharynx, and larynx, as described in one of its titles: *progressive labio-glosso-pharyngeal and laryngeal paralysis*.

The degeneration of the hypoglossal nucleus accounts for the tongue symptoms, the affected accessory portion of the eleventh and vagal nuclei causing the pharyngeal and laryngeal troubles. The one difficulty is to account for the affection of the lips without assuming degeneration of the facial nucleus, of which there is often no evidence, and the solution appears to be that those fibres which supply the orbicularis oris, though running in the facial trunk, in reality have their origin from the upper end of the hypoglossal nucleus, and therefore share in the degenerative changes of the latter.

In a typical example the gradual degenerative changes in the hypoglossal nucleus first show themselves by some weakness in the more delicate movements of the tongue, so that certain letters which require a firm muscle for their articulation cannot be clearly pronounced. Some such letters are l, n, r, s, t. As the change progresses, the tongue wastes and becomes more and more powerless, until at last it cannot be pushed out or thrust into either cheek, and finally it lies helpless on the floor of the mouth with its loosely fitting mucous membrane wrinkled over it. At the same time the lips, formed by the orbicularis oris, which derives its nerve supply from the upper part of the hypoglossal nucleus, also show signs of weakness and wasting. At first certain letters, such as o, u, p,

b, m, requiring the lips to be pressed closely together for their formation, are blurred, and later the lips become thin, wasted, and so weak that they can no longer be approximated, the saliva constantly dribbling from the mouth. It is the drooping of the lower lip that helps so much to give the characteristic facial expression to these cases.

The degeneration of the tenth and eleventh nuclei causes various degrees of difficulty in swallowing. Fluids regurgitate through the nose, and the voice becomes "nasal" owing to deficient movement of the soft palate incompletely separating the oral from the nasal cavity, while the loss of the pharyngeal reflex is a source of danger by allowing the food to pass into the larynx.

When the disease begins in the lumbar region, symptoms similar to those described for the arms are first noticed in the feet and legs, and in the rarer instances where the nuclei of the third, fourth, and sixth cranial nerves degenerate, a loss in the corresponding ocular movements will be seen.

So far, we have considered the effects of the changes in the cells of the lower motor neurons. But while the degeneration of the cells in the anterior horns of the cervical region of the cord is proceeding, some degeneration is frequently also taking place in the fibres of the lateral columns of the cord, as is shown clinically by increased knee-jerks, extensor response of the plantar reflex, and some degree of weakness and spasticity of the lower extremities.

This degeneration of the lateral columns is secondary to changes in the motor cells of the upper neuron (*i.e.*, in the cells of the motor area of the cortex), to which allusion has already been made. The effects of such upper neuron changes are, of course, masked where those due to lower neuron degeneration are present, but as in the

majority of cases of progressive muscular atrophy the lower neuron changes are for a considerable time limited to the arms, the legs are free to show the signs of upper neuron degeneration. Hence, in cases of progressive muscular atrophy, wasting of the arms is commonly found combined with some degree—often very slight—of spasticity in the legs, together with the corresponding changes in the reflexes, viz., increased knee-jerks and extensor response of the plantar reflex.

This association of symptoms leads us a step further, for when these spastic signs in the legs are well marked the condition is known as amyotrophic lateral sclerosis (i.e., a progressive muscular atrophy of the arms and a lateral sclerosis of the legs). No sharp distinction can then be drawn between progressive muscular atrophy and amyotrophic lateral sclerosis, for in so many cases of progressive muscular atrophy there is, in addition to degeneration of the cells of the anterior horns, some degeneration of the lateral columns, and it is according to the degree of this degeneration that the case assumes the picture of progressive muscular atrophy or of amyotrophic sclerosis. Some authors deny that there is any separate condition which should be known as amyotrophic lateral sclerosis, and argue that since most cases of progressive muscular atrophy show some signs of lateral sclerosis, the separation is an artificial one. Others, although recognising the frequency of the presence of some degree of lateral sclerosis in progressive muscular atrophy, prefer to reserve the title of amyotrophic lateral sclerosis for the cases in which the degeneration of the lateral columns is a prominent and progressive feature of the disease. While every gradation can doubtless be recognised, the two terms are useful clinically to indicate whether the upper neurons have or not undergone any material degree of degeneration.

Progressive bulbar paralysis, though really a

part of progressive muscular atrophy, must similarly be retained as a useful clinical term.

Diagnosis of spinal forms.—When the degeneration begins in the nuclei of the cord the wasting indicates at once that the disease is in the lower neuron. This narrows the issue down to lesions of the anterior horns, the peripheral nerves, and the muscles themselves.

Peripheral neuritis must be excluded by the absence of (1) pain, (2) anæsthesia, and (3) the reaction of degeneration, and the grounds for decision will be further strengthened if the area of wasting tends to correspond to the distribution of motor roots and not to that of peripheral nerves.

Myopathies.—Some of the myopathies are difficult to distinguish from progressive muscular atrophy, especially when they commence in the muscles attached to the shoulder-girdle, but when they occur in young people about or before the age of twenty, and when they attack the facial muscles round the angle of the mouth, their nature is generally clear.

Syringo-myelia.—Syringo-myelia closely resembles progressive muscular atrophy when it commences with wasting of the hand muscles. Its nature is demonstrated by the loss of sensation for pain and temperature, while that for touch is generally unimpaired.

When the upper neuron is also extensively degenerated, the wasting in the arms is often combined with spastic paralysis of the legs, and the disease then merges into that of *amyotrophic lateral sclerosis*, from which it can never be very sharply distinguished.

Diagnosis of bulbar forms.—When the degeneration begins in the nuclei of the medulla, the principal conditions it might be confused with are those arising from diphtheria and myasthenia gravis.

In post-diphtheritic paralysis the symptoms are

frequently those of bulbar distribution, *e.g.*, difficulty in swallowing and regurgitation of fluid through the nose, but the rapid onset is unlike the progressive form. The history of an antecedent attack of diphtheria makes the diagnosis plainer, and in the absence of this the subsequent history of the post-diphtheritic paralysis soon clears the matter up. Exceptionally, as mentioned on page 355, the effects of the diphtheritic poison are more permanent; thus, on seeing the patient for the first time, an erroneous diagnosis might be made, but the non-progressive character of the lesion would probably arouse suspicion as to the real nature of the condition.

In *myasthenia gravis* the symptoms vary from time to time, and moreover there is seldom any marked degree of wasting. The myasthenic reaction, if present, is diagnostic.

Vascular lesions in the bulb (especially thrombosis) will cause paralytic symptoms if they occur in the neighbourhood of the nuclei. Their acute onset and more variable course will serve as distinguishing features.

Acute inflammation of the bulbar nuclei, analogous probably to those of acute anterior poliomyelitis, has been described, but this is very rarely limited to the bulb.

Diagnosis of the ocular forms.—In those rare instances where the nuclei of the eye muscles are the first to degenerate, ocular palsies due to tabes, myasthenia gravis, syphilis, and cerebral tumours have to be excluded.

Prognosis.—The course which progressive muscular atrophy usually runs is sufficiently implied by its name.

Extension to the trunk or bulb is generally only a matter of two or three years at the most, and after either event the patient's life must always be precarious. The dangers from bulbar paralysis are choking and broncho-pneumonia due

to particles of food passing into the lungs, and an attack of bronchitis is very apt to cause death when the trunk muscles of respiration are wasted.

In the absence of any knowledge of the toxin it is difficult to say whether progressive muscular atrophy is ever arrested, but it is certain that symptoms which for a time are indistinguishable from progressive muscular atrophy sometimes cease to progress. It may be that in such cases there is a poison which produces local changes in some of the motor cells. In others the wasting perhaps depends upon a lesion confined to the periphery of a nerve, though the electrical reactions are often opposed to such a view.

Anyhow, whatever may be the exact pathology of such cases, a knowledge of the possibility of their existence will sometimes prevent an erroneous diagnosis and consequent prognosis of too severe a character.

Treatment.—Until the nature of the cause of the degeneration is known, efforts have to be made to counteract its effects by keeping up the nutrition of the nerves and muscles.

With regard to drugs, strychnine, iron, cod-liver oil, and other tonics are all useful. Strychnine, given regularly, seems sometimes to check the extension of degeneration, and it is thought to be more efficient when it is given under the skin. When there is much spasticity, strychnine should not be given.

Massage and electrical treatment in moderation help to keep up the strength of the muscles.

When bulbar symptoms are present the patient must be fed carefully to prevent food from going into the air-passages. In bad cases a tube may be required.

CHAPTER XVI

SPASTIC PARAPLEGIA; AMYOTROPHIC LATERAL SCLEROSIS; ATAXIC PARAPLEGIA

SPASTIC PARAPLEGIA

PRIMARY spastic paraplegia, or lateral sclerosis, is an example of an uncomplicated lesion of the upper motor neurons, and is characterised by a degeneration of only those fibres which compose the lateral tracts.

Etiology.—This degeneration appears to be the result of nutritional changes of the motor cells in the cerebral cortex from which the fibres arise, but to what these alterations in nutrition are due is uncertain. Syphilis, injury, lead poisoning, and the toxins of acute infective disease have all been assigned as causes.

In the majority of cases the disease is first noticed between the ages of 20 and 40.

Symptoms.—The main features of this disease are progressive weakness and rigidity, which first show themselves in the lower extremities. The patient notices that he cannot walk so far as he was formerly able, and that, after resting, the limbs feel stiff upon using them again; this stiffness is more especially noticed when he first gets up in the morning. As the symptoms get worse he seeks advice, and it is then that the signs of degeneration of the upper neurons are detected. Loss of power, rigidity, increased reflexes, and the extensor plantar response are present, but there is neither obvious wasting nor are there changes in

the electrical reactions of the muscles. There is no loss of sensation or sphincter trouble, and there are, in fact, no other signs to be found than those which can be accounted for by degeneration of the lateral columns.

As a rule the symptoms get gradually, though slowly, worse, until at last walking becomes difficult or impossible. The rigidity is always a marked feature, and the flexors tend to overcome the extensors, just as the adductors overpower the abductors. Sometimes the joints, when moved beyond a certain distance, spring into the position of flexion or extension; from this the term "clasp-knife" rigidity arises.

The gait is that known as "spastic," and is chiefly dominated by the rigidity which prevents the feet from being freely raised. At the same time, owing mainly to weakness of the flexors of the ankle joints, the toes are dragged along the ground. Hence the gait is stiff, and the feet are shuffled along the ground. Cramps and spasmodic twitchings of the legs are often complained of.

Diagnosis.—The steps taken in the diagnosis of a case of lateral sclerosis will be somewhat as follows.

The patient will probably come complaining of loss of power and stiffness, the existence of which will be confirmed. On testing the deep reflexes, the knee-jerks and the tendo Achillis jerks will be found exaggerated, as also may be the tendon reflexes of the arm. The plantar reflexes will be active, and the big toe will turn deliberately upwards (the extensor response).

This combination of loss of power, spasticity, increased deep reflexes, and an extensor response warrants an inference that the lateral columns are at fault, and further evidence of a negative kind in support of this inference will be found in the absence of wasting and of electrical changes in the muscles.

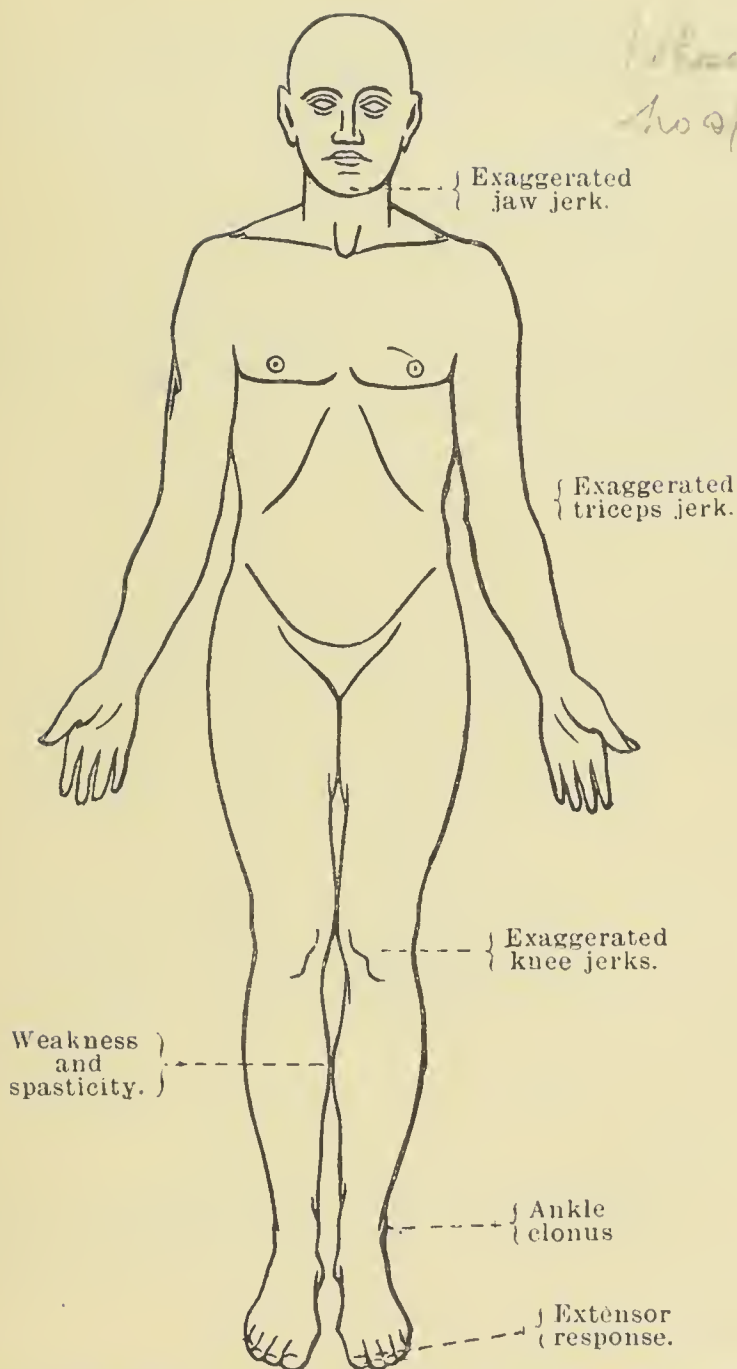


Fig. 42.—Summary of the principal symptoms in spastic paraplegia.

Signs may also be found in the arms showing that the degeneration is going on at that level. If nothing else be found, we are face to face with degeneration of the lateral columns only, and, always bearing in mind the possibility of the clinical picture changing with time into one of disseminated sclerosis, or of the revelation of some gross lesion which may for a time be indiscernible, the diagnosis of primary spastic paraplegia may be made. Primary lateral sclerosis uncomplicated by lesions of other columns is a rare condition; indeed, some authors have gone so far as to doubt its existence, and incline to the belief that all cases which at first appear to be of this kind subsequently develop other symptoms which show that the degeneration is in reality more widely spread. This doubt is largely justified by the scanty pathological evidence that can be brought to show that the degeneration remains confined to the lateral columns throughout the whole course of the disease.

On the other hand, degeneration of the fibres of the lateral columns occurs frequently in association with degeneration of the motor cells of the cord, giving rise to the combination of symptoms known as amyotrophic lateral sclerosis, and therefore there appears to be no reason why in certain cases the fibres of the lateral columns should not degenerate independently of those of the lower neuron, and give rise to primary lateral sclerosis.

In any case, the greatest care must be taken to exclude the possibility of an extending lesion before concluding that the disease is a paraplegia due to primary degeneration of the fibres of the lateral columns.

A large number of cases, in which at the time of examination the only parts apparently affected are the lateral columns, prove later to have been in reality cases of disseminated sclerosis in the

early stages, as is shown by the subsequent appearance of such signs as nystagmus, scanning speech, ocular palsies, and loss of control over the sphincters.

Spastic paraplegia may be secondary to many other conditions, among which spinal caries, tumours, myelitis, and injury of the spine may be mentioned, and it may happen that in any of these the spastic symptoms are for a time sufficiently predominant to make it appear that the lateral columns are alone diseased. When, for instance, in spinal caries, the cord is compressed by inflammatory and granulation tissue, there are often, for a long time, no signs of impaired conduction of sensory impulses, although the posterior columns are naturally injured.

The functions of motor fibres are the more easily disturbed, and thus signs of disease of the lateral columns appear first, and, unless the back be carefully examined, the case may be passed as one of primary spastic paraplegia.

Implication of the posterior columns (*ataxic paraplegia*) will be recognised by the presence of ataxy.

Lastly, spastic paraplegia may be closely simulated by *hysterical paraplegia*.

In organic disease almost every kind of symptom may in its turn be absent from any particular case, but when present such symptoms do not contradict one another, as they so frequently do in hysteria. In lateral sclerosis the signs that are present should be compatible with each other. For instance, the degree of sclerosis which gives rise to an increase of the knee-jerk should also give an extensor reaction of the plantar reflex, and therefore when, in paraplegia, an exaggerated knee-jerk occurs in company with a permanent flexor response of the plantar reflex, it is a contradiction of signs which should at any rate arouse suspicion. Ankle clonus is useful when it is well obtained, as

a sign of organic disease. In functional disease the clonus, when it occurs, is generally feebly sustained, and ceases when the foot is well pressed home. However, this type, known as pseudo-clonus, often occurs during different phases of organic disease.

In the gait of functional disease the whole leg is dragged in a peculiar manner, and there are generally other signs, such as anæsthesia, and variations in symptoms which are not compatible with primary lateral sclerosis. Further, a sudden onset is against primary lateral sclerosis, which comes on very gradually.

Pathology.—The characteristic changes are those produced by a degeneration of the lateral columns, but it is rare to find a spinal cord in which these alone are affected. The degeneration probably begins in the nerve fibres themselves, and the overgrowth of neuroglia is secondary. Probably the cause, as already stated, is failing nutrition of the cortical cells, on which the nutrition of the fibres depends.

Prognosis.—Degeneration limited to the lateral columns is not a direct danger to life. The changes progress slowly, and last over an indefinite number of years. Sometimes the degeneration appears to be arrested, but there is no possibility of cure in the sense of restoration to the previous condition of health.

Treatment.—No methods of ensuring arrest of the primary form are known, but general tonics must be given. Strychnine is often unsuitable because it increases the rigidity.

The general principles of treating rigidity (p. 30) are applicable here.

In many of the secondary forms the possibility of removing the cause of the degeneration is, of course, much greater.

Where the trouble appears to be due to a tumour which, after fair trial, does not yield to

treatment by mercury and iodides, the desirability of calling in the aid of surgery must be considered.

In caries of the spine (Pott's disease) the pressure is usually produced, not by the displaced vertebræ, but by the inflammatory and granulation tissue which springs up round the cord. This adventitious tissue can in favourable circumstances be absorbed, and extraordinary improvement in the nervous system often follows a prolonged and absolute rest, which may be combined with extension by weights to the feet. Cod-liver oil, iodide of iron, and other methods of improving general nutrition are all helpful.

When complete rest is prescribed for caries it is generally advisable to continue it for, say, from three to six months. If improvement occurs, fixation of the spine may be still continued by a well-fitting poroplastic jacket. On the other hand, if the signs show no abatement, the question of laminectomy should be considered. In some instances great improvement has followed this latter procedure, but in others the presence of a secondary myelitis mars the possibility of any full return of power; and it is in no sense an operation to be lightly recommended in this class of case.

AMYOTROPHIC LATERAL SCLEROSIS

In amyotrophic lateral sclerosis, degeneration of both upper and lower motor neurons takes place at the same time. Clinically, the symptoms are a combination of those of lateral sclerosis and progressive muscular atrophy, and reference has already been made to the relations between amyotrophic lateral sclerosis and the last-named disease.

Etiology.—Nothing definite is known concerning the cause of the degeneration. Presumably it is of the same nature as that of progressive muscular atrophy, but at present no definite statement as to its cause can be made.

exposure to cold + wet — fatigue
 nervous system
 Age. 25. 50 — Both sexes

Pathology.—The essential cause of the degeneration is failure of the nutrition of the motor cells of the cerebral cortex and of the anterior horns of the cord. Sections of the cord show sclerosis of the pyramidal tracts and wasting of the motor cells of the anterior horns, while in the brain some degeneration of the motor cells of the cortex can be demonstrated.

Symptoms.—The symptoms vary according to the extent of the wasting and spasticity, but in a typical case the picture is one of lateral sclerosis together with wasting of the muscles of the upper limb. The onset in such a case is insidious, and usually resembles that of primary lateral sclerosis, in which weakness and stiffness of the legs are the early signs; but in some cases wasting of the hands is the first sign to attract attention, and then the onset more closely resembles that of progressive muscular atrophy.

The signs of sclerosis of the pyramidal tracts are generally very obvious in amyotrophic lateral sclerosis, as shown by the exaggeration of all the tendon reflexes and the presence of clonus, which can often be obtained in the arms and jaw as well as in the legs. At the same time some wasting can be noticed to be taking place in the muscles of the hands or arms.

In other cases the wasting is, at any rate for a time, the predominant feature; the rigidity and weakness are slight, and the clinical picture is then more nearly like that of progressive muscular atrophy than of lateral sclerosis.

Diagnosis.—The diagnosis of amyotrophic lateral sclerosis is made from evidence of the co-existence of sclerosis of the lateral columns and degeneration of cells of the anterior horns of the cord. It thus forms a link between primary lateral sclerosis and progressive muscular atrophy, and, indeed, there are few cases of progressive muscular atrophy in which some signs of degeneration

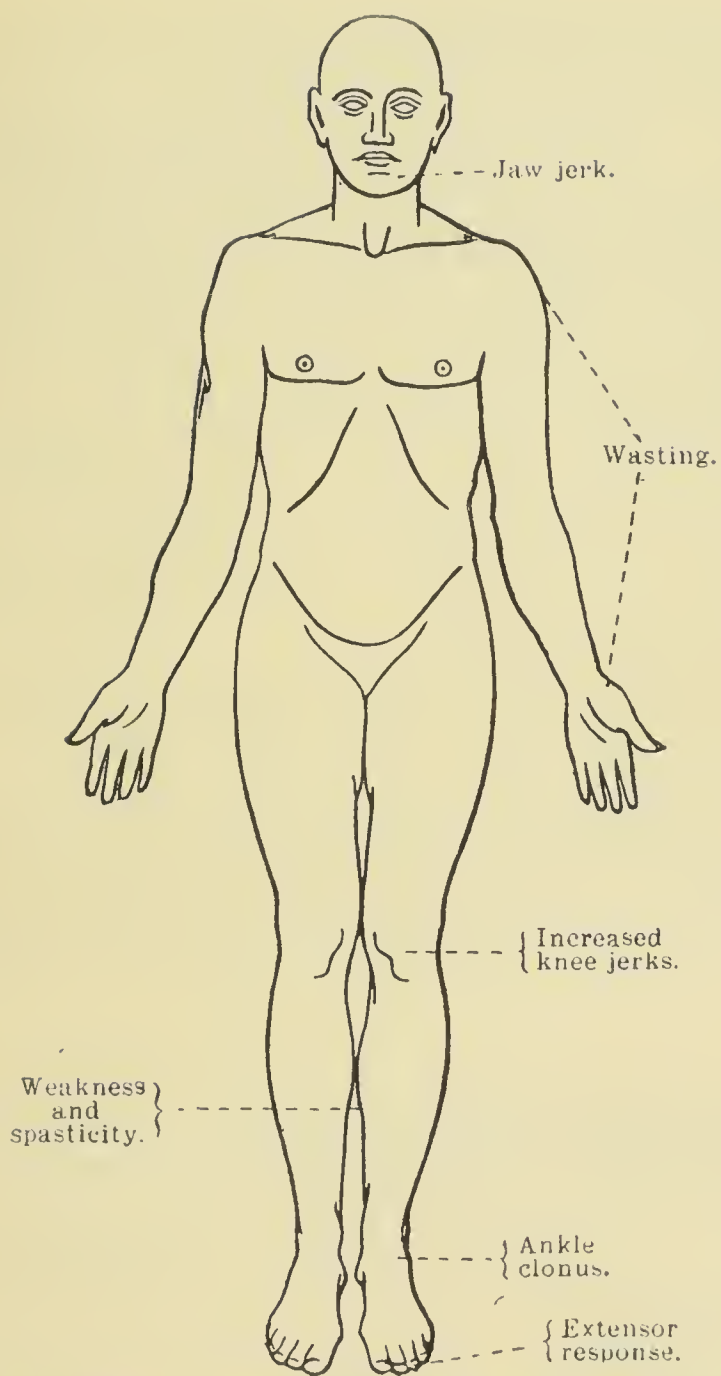


Fig. 43.—Summary of the principal symptoms in amyotrophic lateral sclerosis.

of the lateral columns cannot be found. Whether all such cases should be included in the category of amyotrophic lateral sclerosis is largely a matter of individual opinion. It is on the whole, perhaps, best to speak of such cases as progressive muscular atrophy associated with signs of commencing degeneration of the upper motor neuron, and to reserve the term amyotrophic lateral sclerosis for those in which the spasticity is a predominant feature from the beginning, but no sharp definition can be drawn, since cases of every gradation are met with. When the symptoms arise insidiously the nature of the disease may for a time be overlooked, and the association of the flabby muscles of the arms with stiffness and weakness of the hands may be attributed to *rheumatoid arthritis*.

Prognosis.—Amyotrophic lateral sclerosis runs much the same course as progressive muscular atrophy, and there are the same dangers to life in both diseases.

Treatment.—What has already been said of progressive muscular atrophy is applicable here, with the exception that strychnine may have to be withheld on account of its tendency to increase the rigidity.

ATAXIC PARAPLEGIA

Ataxic paraplegia is due to sclerosis of the lateral and posterior columns. As in primary lateral sclerosis, many of the cases which, at one time, appear to be examples of ataxic paraplegia develop other symptoms later, thus showing that the degeneration has not been confined to these two columns.

Etiology.—What has been said about the causation of primary spastic paraplegia is applicable here, with the additional statement that toxins associated with extreme anæmias, diabetes, and other debilitating diseases seem to cause com-

bined sclerosis rather than one which is limited to the lateral columns. Ergotism from eating bad grain and pellagra from diseased maize may, among other symptoms, cause sclerosis of the posterior and lateral columns of the cord.

Symptoms.—The symptoms come on gradually, with weakness and spasticity of the lower limbs, just as in lateral sclerosis, and to these a variable degree of ataxy is added. Thus the clinical picture is one of lateral sclerosis with ataxy. In some cases there is a little difficulty in controlling the sphincters, and in some, sensation is blunted.

Prognosis.—The disease runs a very chronic course, and, unless the sclerosis extends beyond the lateral and posterior columns, does not usually endanger life.

Pathology.—The spinal cord shows sclerosis of the lateral and posterior columns, though in many cases the disease has passed outside their boundaries. In the posterior columns the posterior external column in the neighbourhood of the posterior root zone escapes, in which it differs from the lesion of tabes. The reflex arc is thus not broken, and the sclerosis of the lateral columns accounts for the increased tendon reflexes.

Diagnosis.—The diagnosis must generally be provisional in view of the possible development of other symptoms. As in the case of primary spastic paraplegia, a large number prove later to be examples of disseminated sclerosis, as shown by the occurrence of such symptoms as nystagmus, ocular palsies, and pallor of the optic discs.

Degenerative changes in the cord after a myelitis will cause symptoms that are practically indistinguishable from those of ataxic paraplegia.

Treatment.—No adequate method of arresting the degeneration is known. Treatment must be confined to attempting to improve the general health.

CHAPTER XVII

TABES DORSALIS (LOCOMOTOR ATAXY)

Etiology.—The symptoms of locomotor ataxy are due in the main to a degeneration of the lowest sensory neurons.

But although the degeneration mainly attacks the sensory neurons, some signs of its extension to those of motor function also are often seen, more especially in connection with the ocular nerves, and occasionally, though less frequently, with motor nerves in other parts of the body.

The disease is found in men about ten times as often as in women, and the onset in most cases occurs somewhere between the ages of twenty and fifty.

Occasionally patients with the disease are seen while still in their teens, and to these cases the term juvenile tabes is applied.

The exact nature of the poison is still a matter of doubt, but it is generally conceded that a previous attack of syphilis has an important bearing on the commencement of the degeneration.

A very large proportion of cases of locomotor ataxy have certainly had syphilis—according to some statistics as many as 90 per cent.—and it seems that the syphilitic virus lowers the vitality of the neurons in such a manner that they die prematurely, just as the introduction of some harmful substance into the soil may cause the death of a plant, no matter how favourable may be the climate and other surroundings.

In other instances the vitality of the neurons appears to be depressed to a certain degree only, and their functions, while still performed in favourable circumstances, easily fail on the addition of any extra strain, such as may be produced by excess of alcohol, infective processes, injury, worry, and so forth.

The possible importance of infections other than that of syphilis has recently been brought forward by Ford Robertson, who considers that the essential cause of the nerve degeneration in locomotor ataxy and general paralysis of the insane (for the nature of the pathology of both is probably identical) is a toxin arising from a bacillus closely resembling that of diphtheria, which he has found in the alimentary canal and other tissues of the body.

The reason for the definitely selective action of the toxin on the lowest sensory neurons has been much debated. It is, of course, well known that poisons like lead, diphtheria, alcohol, and arsenic all exercise a selective action on certain groups of fibres, and it may be that the poison which causes tabes acts in a similar manner.

One view is that the poison injures the cell bodies from which the fibres arise and receive their nutrition. In this instance the cell bodies are situated in the posterior root ganglia, and changes in them have been described, although they do not appear to be demonstrable in every case. Moreover, if failure of the nutrition of these cells causes degeneration of the central process of the T-shaped fibres which spring from them, one would expect the peripheral process to degenerate as well, but this does not by any means always occur. Changes have, however, been described in the muscle spindles in which the periphery of some of the nerves terminate, and it may be that these are the first indication of a peripheral degeneration (Batten).

There are anatomical reasons which seem to make the fibres of the posterior nerve roots comparatively less resistant. There is, for example, some slight natural constriction of the roots as they pass through the pia mater, which has been thought to lower their resisting powers, and the arrangement of the lymphatics in relation to the posterior roots is, according to some authors, the reason which makes these fibres especially liable to be affected by toxins.

Orr and Rows* have recently published the results of some important researches on this subject, which show that "in peripheral nerves, spinal roots, and cranial nerves there is a constant stream of lymph ascending towards the central nervous system whose main current lies in the inner meshes or lymph spaces of the fibrous perineural sheath." Toxins reaching the spinal cord and brain by this channel are particularly apt to affect the nerves where they have lost their neurilemma sheath, which spot, Orr and Rows point out, is not quite identical with the place at which the nerves undergo the slight constriction as they pass into the cord.

Lastly, some neurologists consider that the degeneration is produced by a thickening of the spinal membranes which compresses the roots as they pass through the pia mater, i.e., at the point at which they are naturally rather narrowed, and that this thickening is caused by a tertiary syphilitic process. That some thickening of the membranes is frequently present is undisputed, but it is difficult to conceive it as the main cause of the degeneration. If it were so one would expect the anterior roots to suffer more frequently; and, further, there is also the difficulty in accounting for the ocular and other cranial nerve symptoms on this hypothesis.

To sum up, there are at present in vogue three

* *Journ. of Neurology and Psychiatry*, May, 1907.

main hypotheses concerning the cause of locomotor ataxy. They are—

- (1) That *syphilis* is in the majority of cases the essential cause of the degeneration, but that it acts indirectly by lowering the vitality of the nerve fibres, and not through any of the processes which are recognised as belonging to the secondary or tertiary stages. According to this view tabes is spoken of as a disease of "parasyphilitic" origin, and the probable seat of the primary alteration in nutrition is placed in the cells of the posterior root ganglia.
- (2) That the degeneration is the consequence of *syphilitic thickening* of the membranes round the posterior roots, by which the nerve fibres are constricted and destroyed.
- (3) That the degeneration is due to direct infection of the products of a bacillus closely resembling the diphtheria bacillus of Klebs-Löffler.

Acceptance of the third hypothesis is not, of course, necessarily antagonistic to the truth of the first, for it can easily be understood that syphilis may prepare the way for the invasion of other toxins, and indeed there is very strong evidence that such is the case.

Pathology.—It is generally agreed that the pathological process is one of primary degeneration of the nerve fibres, to which the overgrowth of neuroglia is secondary, and this view receives confirmation from the fact that the only degenerated fibres traceable in the cord during the early stages of the disease are those which are continuous with the roots (*i.e.*, exogenous fibres), and not those which pass from one part of the cord to another (*i.e.*, endogenous fibres). Were the primary

condition an overgrowth of neuroglia, no such distinction would be shown between the different sets of fibres.

Of these fibres from the posterior roots which are so prone to degenerate, some proceed up the posterior columns, some complete the reflex arcs by communicating with the cells of the anterior horns, and some terminate in Clarke's column, from which fibres arise to pass up the direct cerebellar tracts (Fig. 44, A, B, and C).

Degeneration is most prominently marked in connection with those fibres which pass up the posterior columns of the cord, and if the course of a single degenerated fibre be traced it will be observed to pass first up the posterior external column (Burdach), and subsequently to find its way into the posterior internal column (Goll), and then to pass up to the medulla, where it terminates.

Thus a section of the cord shows degeneration of the posterior columns, and, since the roots in the lower part of the cord are generally the most affected, the degeneration is there most dense in the posterior external columns, and gradually becomes more marked in the posterior internal columns as it ascends the cord.

Consideration of the mechanism by which symptoms are produced.—The symptoms of locomotor ataxy are the result of degeneration of fibres in the posterior nerve roots. Consideration of the functions of these fibres and of the consequences following their destruction will give a good idea of the different symptoms that occur.

A posterior nerve root is composed of: (1) fibres carrying impulses for sensations of touch, pain, and temperature; (2) fibres along which impressions pass from the muscles, ligaments, and joints; (3) fibres which complete the reflex arcs by communicating with the cells in the anterior horns.

(1) Failure of nutrition of the fibres which convey impressions of touch, pain, and temperature results in anæsthesia over the areas of skin corresponding to the particular roots affected; but since all the fibres in the root are not damaged to an equal extent, until the root is entirely destroyed the anæsthesia occurs in patches, some part of a given root-area being more anæsthetic than others.

Further, one form of sensation may suffer more than another, so that the patches of anæsthesia to touch, pain, and temperature do not necessarily coincide.

This degeneration is accompanied by signs of irritation of the nerves which takes the form of lightning pains and visceral crises.

The loss of recognition of sensory impulses is closely associated with modifications of nutrition in the skin, bones, and joints, constituting the so-called "trophic" changes.

(2) The degeneration of fibres which should convey impulses from the muscles, ligaments, and joints is accompanied by incoordination and a failure to recognise the relative positions in which various parts of the limbs are placed (i.e., loss of sense of position). The power of distinguishing the form of objects is also frequently diminished (astereognosis).

(3) Degeneration of the fibres which communicate with the cells of the anterior horns leads to loss of reflexes and to loss of tone of the muscles. The impulses which constantly flow into the cord

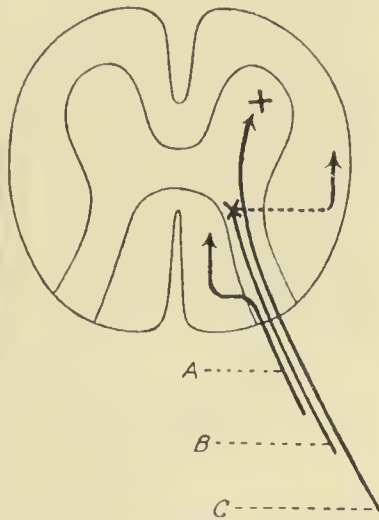


Fig. 44.—Diagram showing the main destinations of fibres of the posterior nerve roots.

through the posterior roots exercise a definite influence upon those flowing out by the anterior roots, and if they are cut off either by experiment



Fig. 45.—Case showing increased range of movement due to hypotonus of the muscles.

or disease, as they are in the case of locomotor ataxy, the "tone" or elasticity of the muscles of the corresponding arc is diminished, and the condition known as "hypotonus" arises (Fig. 45).

If these principles are applied to different

levels of the cord the mechanism of the main symptoms is clearly demonstrated.

Thus from degeneration of the lumbar and sacral roots there arise pains in the legs, patches of anæsthesia, numbness and a feeling as though walking on cotton-wool, trophic symptoms, inco-ordination, loss of the knee and tendo-Achillis jerks, impairment of the action of the sphincter reflexes, and hypotonus.

In the dorsal region the degenerating fibres are apt to cause girdle pains and a zone of altered sensation in the corresponding course of distribution of the roots.

Higher still—in the cervical region—there are signs in the arms corresponding in character to those already enumerated for the legs; and at a still higher level the reflex arc for light is broken and the optic nerve atrophies. VI

Such, then, is the general mode of production of the sensory symptoms of locomotor ataxy, over and above which there may be many others referable to alterations in the motor neurons.

General clinical course.—It will be clear from what has already been said that there can be no constancy concerning the order in which the different symptoms make their appearance.

The onset is generally associated with pains, which, as might be expected, may be felt for a long time, even for years, before the attention of the patient is called to any other symptom, though it must be admitted that a systematic examination would probably often reveal the existence of other signs at an earlier date than they are otherwise recognised. The pains are of a lightning or shooting character, and vary greatly in their intensity and duration, often disappearing for weeks together, to return subsequently for several days at a time. Associated with these there may be hyperæsthesia of such intensity that the patient cannot bear the slightest touch. At first these

pains are almost invariably attributed to neuralgia or rheumatism, or, if they remain for a time confined to the course of a particular nerve, they are liable to be attributed to a localised neuritis, *e.g.*, of the sciatic or ulnar nerve.

In addition to these lightning pains which are felt in the more superficial structures, there may be deeper "boring" pains in the bones or other organs, which often remain for a long time localised to one spot. Cramps in the muscles are also sometimes a prominent feature.

Since the lumbar and sacral roots are generally among the first to degenerate, the knee-jerks are generally lost or much diminished quite early in the course of the disease, and at the same time changes are very frequently occurring at the other end of the system which result in loss of the light reflex.

Thus the combination of symptoms most commonly found early in the disease consists of lightning pains, loss of knee-jerks, and the Argyll-Robertson pupils, *i.e.*, pupils which contract during accommodation but do not react to light, and which may be pin-point or unequal in size.

In addition, if carefully looked for, there is a likelihood of some degree of ataxy being recognised in the lower extremities: but other symptoms often exist for a long time before ataxy is noticed, so much so that a "preataxic stage" of the disease is clinically recognised.

Another early, and not unfrequently the first, symptom is that of primary atrophy of the optic nerves.

The clinical course of the disease may best be described by following the development of the chief symptoms in a typical case.

If the patient comes complaining of "rheumatic" pains, which on inquiry prove to be of a lightning character, and examination shows loss of knee-jerks and the presence of the Argyll-Robertson

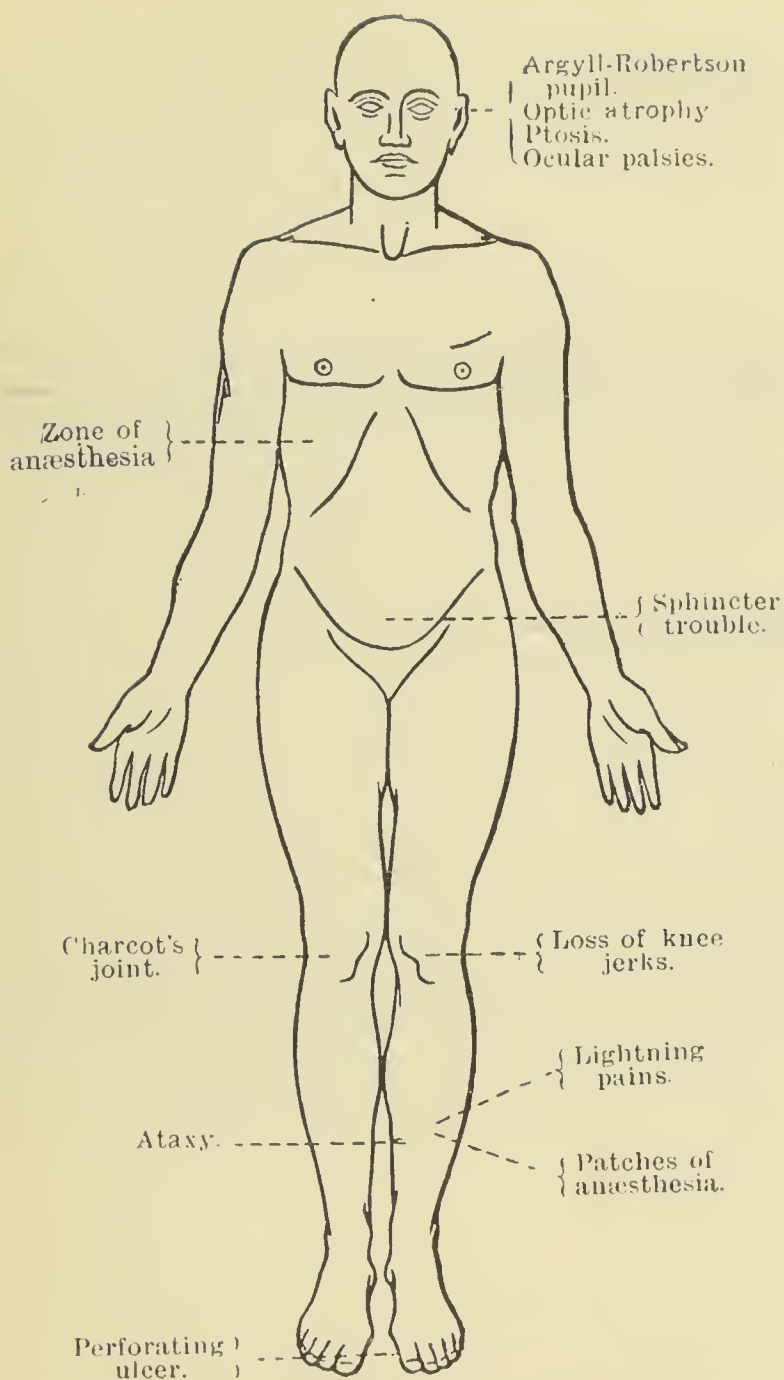


Fig. 46.—Summary of the principal symptoms of tabes dorsalis.

pupil, the diagnosis of tabes in the "preataxic" stage will probably be made.

In such a case other signs will in all probability gradually show themselves, although under favourable circumstances the progress may be very slow or may even become arrested.

If degeneration progresses, the patient's attention will sooner or later become drawn to his unsteadiness both when walking and standing, this unsteadiness being greater in the dark or on such occasions as he may have his eyes shut, e.g., when he is washing his face. Before, however, the patient recognises any difficulty in this direction it can generally be discovered by making him stand with his feet together and his eyes closed (Romberg's sign), or by asking him to try to execute such balancing movements as are necessary for walking with the heel of one foot placed at each step in front of the toes of the other—a very delicate test if performed with the eyes closed.

Various defects in the sensations of touch, pain, and temperature will be found in irregular patches corresponding to the areas supplied by the degenerating nerve roots, and the sole of the foot is a particularly likely place to find such patches. The areas of loss to one kind of sensation do not necessarily correspond to those of another, as is only to be expected when it is remembered that the separate nerve fibres are slowly degenerating independently of one another.

Another sensory disturbance often found quite early in the disease is a zone of analgesia or anæsthesia round the chest or abdomen, indicating that degeneration is proceeding in some of the dorsal roots.

Sexual power is usually greatly diminished or lost by the time the disease is fully developed, but during the early stages it may be temporarily increased.

From this period onwards, if the case be progressive, the ataxy increases in degree, until at last the patient may be quite unable to walk or even to stand. Before, however, this stage is reached many symptoms may or may not develop incidentally and for the onset of which no



Fig. 47 —Perforating ulcer occurring in the course of a case of tabes dorsalis.

order can possibly be given, for, indeed, as already insisted upon, almost any one of all the possible symptoms occurring during the course of the disease may be the first to attract attention.

With this general sketch of the disease it is therefore necessary to consider some of the main symptoms at length.

TROPHIC DISTURBANCES

Perforating ulcers.—Perforating ulcers are characterised by the formation of crateriform indolent sores of various depths, which make but little attempt to heal. The skin at their edges is



Fig. 48.—Charcot's disease of the right knee.

usually thickened and raised to form a dense callosity which often exists as a corn before there is any ulceration (Fig. 47).

The ulcers, which tend to assume a somewhat conical shape, are generally painless, and frequently extend right down to the bone.

Their favourite position is on the fore part of the sole of the foot, but sometimes they appear

on the upper surface of the toes; they show no disposition whatever to occur on the heel.

The underlying factor in the formation of these ulcers is undoubtedly an alteration in the nutrition of the affected parts consequent upon the destruction of sensory fibres, while pressure and injury presumably take a share in the causation. In



Fig. 49.—Charcot's disease of right ankle joint associated with atrophic changes in the bones of the foot.

some cases, as suggested by Tourette, the ulceration may possibly be secondary to diseased bones and joints, and may be in fact a fistula. Under favourable conditions, the most important of which is rest, an ulcer often heals spontaneously, but sometimes first requires such stimulation as is obtained by scraping or by exposure to the X-rays, the latter mode of treatment appearing in some cases to act very favourably.

Osteo-arthropathies.—Changes in the bones and

joints have next to be considered—the so-called osteo-arthropathies.

The joints known as Charcot's joints may occur



Fig. 50.—Atrophy of the head of the humerus associated with a "Charcot's joint." (Middlesex Hospital Museum.)

at practically any period of the disease, and are characterised by a painless and often rapid swelling due to the effusion of fluid, which, after being absorbed, often leaves the joint in a flail-like



Fig. 51.—Skigram showing atrophy of the bones of the left foot. Note the shortening of the great toe.

condition, due partly to the laxity and softening of the ligaments and partly to changes in the articular surfaces of the bones; these last-mentioned changes may all occur without any definite degree of effusion. The knees, elbows, hips, and shoulders are among the joints which most commonly suffer, while another common situation, perhaps not so often recognised, is that of the big-toe joint (Figs. 48 and 49).

The essential process in all these joints is one of atrophy, which is sometimes accompanied by irregular bony outgrowths similar to those associated with rheumatoid arthritis. This form, to which the term hypertrophic is sometimes applied, appears to occur more often in the knee joints than elsewhere, while the shoulder and hip are more likely to show signs of advanced atrophy, as evidenced by dislocation of the head of the bones.

The exact mechanism by which these joint troubles are produced is obscure. The influence of traumatism as a direct cause can be largely excluded by their frequent occurrence in the pre-ataxic stage of the disease, and the possibility of their being of syphilitic origin is negatived by the fact that changes of practically the same nature are to be found in syringo-myelia, concerning which there is no question of the influence of syphilis.

The bones.—In locomotor ataxy the proportion of the lime salts in the bones is frequently reduced, hence the bones become soft and brittle, and “spontaneous” fractures (*i.e.*, fractures brought about by a very slight amount of violence) arise.

The atrophy of bones, especially those of the feet, is often obvious during life. The big toe is very frequently shortened, owing, as shown by the X-rays, to wasting of its bones. In the feet and, more rarely, in the vertebræ the bones and joints are apt to show changes at the same time (Figs. 50, 51 and 52).

THE CRISES

Paroxysmal disturbances of function (known as "crises") are apt to occur in connection with the various viscera. In their production they



Fig. 52.—Skiagram showing very advanced atrophy of the bones of the foot. (From a case under the care of Mr. T. H. Kellock.)

appear to be analogous to the lightning pains, and in some cases they probably depend upon degeneration of fibres of the sympathetic system.

Gastric crises.—These form the commonest variety of crises. Every few weeks the patient has attacks of intense pain and sickness, which for the time being entirely incapacitate him from

work. The duration of these attacks varies from a few days to a week or more, and in the intervals there may be no symptoms whatever.

Wilfred Harris has observed the occasional occurrence of epileptiform attacks during the gastric crises, and considers that there is some causal relationship between the two conditions.

Laryngeal crises.—In these the patient gets signs of spasm of the laryngeal muscles, accompanied by a barking and crowing cough, which in some ways resembles that of whooping-cough.

Other organs subject to crises are the bladder, rectum, and kidneys.

OCULAR SYMPTOMS

The Argyll-Robertson pupil is one of the commonest symptoms of tabes dorsalis. Its characteristic is loss of reaction to light, while contraction during accommodation remains. The pupils are generally very small (pin-point), often unequal, and frequently show some loss of that perfect rotundity by which they are distinguished in health.

The Argyll-Robertson pupil is often the earliest sign of tabes, and it may occasionally be observed to exist for a time in one eye only. When the degeneration is more extensive the contraction during accommodation may also be absent.

in arc. The lesion causing the loss of light reflex in the Argyll-Robertson pupil is most probably situated in the fibres which complete the reflex arc by joining the optic nerve to the nucleus of the third nerve. If a case be taken in which there is no question of optic atrophy, it is obvious that the fibres of the optic nerve are healthy, and in like manner the ability of the pupil to contract in association with accommodation shows that impulses can pass freely along the fibres of the third nerve. The inference, therefore, is that the degeneration in such a case has taken place in the fibres between the two nerves.

Another view is that the Argyll-Robertson pupil is caused by degeneration of the ciliary ganglion.

Optic atrophy.—A primary grey atrophy of the optic nerve is apt to occur in the course of locomotor ataxy, and sometimes failure of sight from this cause is the first symptom to attract the

*degener.
Ciliary Ganglion*

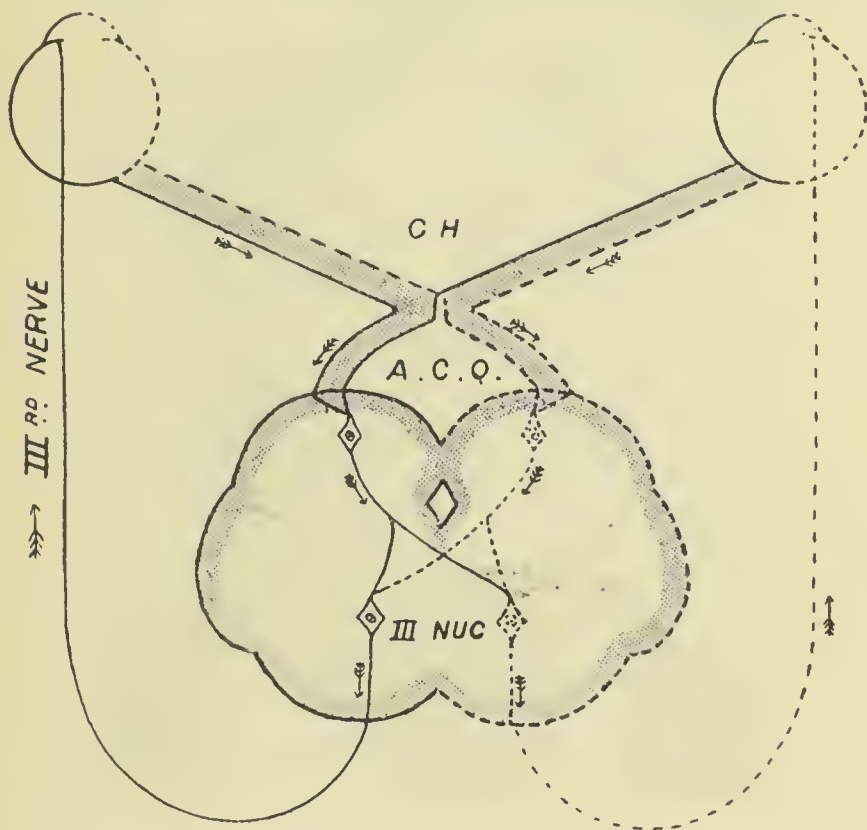


Fig. 53.—Diagram of the pupil reflex to light. (Wilfred Harris.)

patient's attention. The disc assumes a pearly-white appearance, and the vessels running over it are relatively small. The rate at which sight is lost varies greatly, and it may be some three, four, five, or more years before anything like complete blindness supervenes.

In most cases the loss of the light reflexes and atrophy of the optic nerve are both due to

degeneration of sensory fibres, and are thus analogous to the degenerations which take place in the posterior roots of the spinal nerves.

Paresis of muscles.—Paresis of the ocular muscles indicates a lesion of the motor neurons,



Fig. 54.—Case of tabetic ptosis, showing the compensatory contraction of the occipito-frontalis.

and it is in this region that the motor symptoms in tabes are most commonly found.

Any of the ocular muscles may be affected, but the external rectus is the one most prone to suffer. Squint and diplopia are the result, but as a rule the paresis is only of temporary duration, and it is common for patients to complain from time to time of double vision, which completely passes away in the intervals.

Much less common, but more important, are the permanent paralyses in which all the branches of the third nerve are liable to be affected, and which are probably due to degeneration of the motor nuclei.

Ptosis is sometimes temporary, thus resembling the transient paralysis of other ocular muscles, but at other times it may be permanent and be part of a complete paralysis of the third nerve (Fig. 54).

Other cranial nerves.—No cranial nerves are exempt from the possibility of degeneration; the olfactory and auditory nerves may suffer, and sometimes there are signs of degeneration of the nuclei in the medulla. Anæsthesia and pains not unfrequently occur over the regions supplied by the fifth nerve.

Diagnosis.—In considering the diagnosis of locomotor ataxy it is important to remember the possibility of the signs appearing in almost any order. In one case the pains may be the most prominent symptom; in another a joint may be the chief cause of anxiety; in a third it may be the eyes that first give rise to trouble; and in yet another there may be difficulty in exercising full command over the bladder. It is most necessary in diagnosing tabes to remember that undue stress must not be laid upon the absence of one or more signs that ought to be present in a "typical" case. For instance, because the knee-jerk is usually lost by the time most patients come for diagnosis, its presence, and sometimes even its increase, must not be allowed to negative the possibility of tabes if other signs point to the wisdom of such a conclusion. When the roots are affected in such a variable order as they are in tabes, it is no wonder if those upon which the presence of the knee-jerks depends are not always among the first to degenerate.

The presence of Argyll-Robertson pupils in

association with any other suspicious symptom—e.g., lightning pains, bladder trouble, or a swollen joint of doubtful nature—is practically pathognomonic of tabes, provided that the commencing onset of general paralysis of the insane can be excluded.

It occasionally happens that, during the routine examination of patients, Argyll-Robertson pupils are discovered as isolated signs and are associated with no other detectable morbid conditions whatever. The question naturally then arises as to what interpretation should be put upon such a case. On the whole it would seem that the most reasonable attitude to adopt in such cases is that which looks upon these pupils as evidence of local degeneration, probably of parasyphilitic origin; but experience shows that, although their presence is very suspicious, it is nevertheless possible for them to exist for an indefinite time without any signs of degeneration of the nervous system appearing elsewhere.

Peripheral neuritis.—Cases of peripheral neuritis in which sensory symptoms are prominent sometimes resemble tabes in many respects, for common to the two disorders may be pains, loss of knee-jerks, and incoordination, and occasionally the similarity is further increased by the presence of sores on the feet, which may be mistaken for “perforating ulcers.” The closest similarity seems to occur in some of the cases of neuritis due to alcohol and diabetes, but generally a systematic examination removes the doubt by revealing positive signs of one or other disease.

An Argyll-Robertson pupil, for instance, would be conclusive evidence in favour of tabes, while loss of power and electrical changes would strongly support the supposition of neuritis.

Friedreich's disease has several symptoms in common with tabes e.g., loss of tendon reflexes and incoordination—but the age of onset and the

family nature of the disease, together with the speech defects, nodding movements, and nystagmus, usually serve to distinguish it, though, as far as age is concerned, it must be remembered that cases of juvenile tabes may occur.

Ataxic paraplegia.—The combination of spasticity with incoordination, together with the increased knee-jerks and the absence of eye symptoms, will serve to distinguish ataxic paraplegia from tabes.

Cerebellar disease.—The ataxy of cerebellar disease does not usually become distinctly worse when the eyes are closed.

If there is a tumour the history and symptoms will not be suggestive of tabes, and if the ataxy is due to cerebellar deficiency the history of the case will enable tabes to be excluded.

Disseminated sclerosis.—Disseminated sclerosis appears in so many forms that in some of them it may occasionally resemble tabes, but even in those instances where confusion is likely to arise, exaggerated knee-jerks (loss of knee-jerks is very uncommon in disseminated sclerosis), alteration in speech, nystagmus, and tremors on movement are generally present in some degree.

Spinal syphilis.—Syphilitic disease of the membranes, causing secondary changes in the posterior roots, combined as it may be with thrombosis of vessels in the posterior columns of the cord, may give rise to signs which are in some ways almost indistinguishable from those of tabes.

Pain in the back, loss of power (of flaccid type, due to implication of anterior roots, or of spastic type, due to extension of the thrombotic processes into the lateral columns), and the variability of the symptoms are among the distinguishing features. The presence of co-existing signs of cerebral syphilis often assists in making the diagnosis.

Prognosis.—Probably in no other chronic

disease of the spinal cord does degeneration vary more in its rate of progress than it does in tabes.

After the appearance of the first symptoms, many years often elapse before there are any signs of ataxy. The symptoms may cease to progress at various stages of the disease, and in a certain number of cases some definite improvement occurs. In these circumstances it will be obvious how important it is to try to form an estimate of the probable future of every case in order to be in a position to give some practical advice regarding the best course for the patient to pursue.

This, as far as is possible, may be accomplished by paying attention to the patient's age and circumstances, the rate at which the degeneration has progressed, and the position of the first signs of degeneration.

It has been observed that the course of tabes is, as a rule, much slower when it begins in elderly people of 50 or 60 than when it attacks those of younger years, e.g., from 25 to 30. In these latter the degeneration often proceeds very rapidly.

The circumstances of the patient must be taken into account in forming a prognosis, since those who have money and leisure are in a better position to combat the disease than those who are obliged to continue their daily work.

If at the time of observation the patient gives a history of tabetic symptoms existing over a period of some years, and examination shows that the extent of the degenerative processes is still very limited, it is reasonable to infer, with certain reservations, that the changes will continue to proceed slowly.

In this connection Sir William Gowers has directed attention to a group of cases in which, though lightning pains have existed for a number of years, there is no loss of knee-jerks and no sign of ataxy. According to Sir William Gowers,

these should be considered as a special variety of tabes, depending upon some peculiarity of the toxin, rather than ordinary cases in which development of the disease has been abnormally slow. The future of such cases appears to be comparatively favourable, and until the knee-jerks are lost no ataxy is likely to arise.

It is also well known that patients who develop optic atrophy early often remain for a long time without any signs of ataxy.

In predicting a slow degeneration for the future on the strength of one that has been slow in the past, it must not be forgotten that very rapid increase in the intensity of symptoms is sometimes brought about by shock, illness, exposure to cold, or injuries.

Treatment.—Those who believe tabes to be the direct result of syphilis naturally put their trust in antisyphilitic remedies. They prescribe inunctions, injections, and other courses of mercurial medication, often in conjunction with large doses of potassium iodide. Most neurologists, however, do not see their way to accept the hypothesis that syphilis is the *direct* cause of tabes; nor are they able to confirm the curative action of mercury and iodide. That the patient often feels better for a time after a course of mercurial treatment is true; nor is this surprising, since mercury has a potent effect on metabolism, but it cannot possibly be said that there is any effect which is strictly comparable to that obtained in the ordinary lesions of tertiary syphilis.

In the present state of knowledge it is wise to keep an open mind on the subject, and perhaps to prescribe antispecific remedies for a time in those cases where they have been neglected or inefficiently carried out at the time of infection.

Thanks to the researches of Ford Robertson and his colleagues on the infective origin of general paralysis of the insane and locomotor ataxy, there

is reason to hope that treatment by serum may eventually become a practical therapeutic measure in both these diseases. *R. H. + S. P. 1*

Apart from these remedies, reliance must be placed on increasing the resisting powers of the patient by improving his nutrition, which can best be done by prescribing suitable tonics with a proper amount of food and rest.

Of tonics, the author places most reliance on iron, and it seems reasonable to suppose that an improvement in the quality of the blood will be followed by an improvement in the nutrition of the nerve fibres. The perchloride, in doses of 10 to 15 minims three times a day, appears to answer best, but if it disagrees other preparations may be substituted. If it be thought advisable, arsenic may be given with the iron. Aluminium chloride was introduced into therapeutics by Sir William Gowers for its apparent usefulness in modifying the intensity and frequency of the lightning pains, and it also may be given in combination with the iron and arsenic in some such prescription as the following:—

Tinctura ferri perchloridi	m. xii.
Liquoris arsenici hydrochlorici	m. iii.
Aluminii chloridi	gr. iii.
Spiritus chloroformi	m. xv.
Aquam ad	3 i.

Sig. : Two tablespoonfuls to be taken three times a day after meals.

Of course, to obtain any real benefit the iron must be given regularly for a long time, and in the author's experience there is seldom any difficulty with the digestion if the precautions usually taken in giving iron are observed.

Strychnine is a drug which may also occasionally be given with benefit.

Plenty of nourishing food should be taken, for the nutrition of these patients is frequently poor, and, in many instances, cod-liver oil is useful.

Rest is an important factor in the course of treatment. The degeneration seemingly has a tendency to occur in those nerves whose functions are most hardly used, and to continue to exercise these nerves as much as before is only to court an extension of the degenerative processes.

Walking (for the disease generally begins in the roots at the lower part of the cord) should be slow, the distances should be short, and, whenever possible, driving should be substituted. Fatigue both of mind and body should be avoided as much as possible, and it is scarcely necessary to add that plenty of fresh air, within doors as much as without, is essential.

The various forms of treatment by electricity have, at most, nothing more than a passing tonic effect.

To restore the function of those fibres which are already dead is, of course, impossible, but in suitable circumstances, and by careful attention to details, there is no doubt that a great deal can be done to retard, and even to arrest, the extension of degeneration.

It must also be recollected that in disease, just as in health, function is often a degree above or below what may be the average level for any particular person. Tabetic patients with optic atrophy can see much better on some days than others: so, also, those with ataxy can walk better on some days than others. To keep these patients at their highest possible level of health should be one of the objects of treatment.

Treatment of lightning pains.—To relieve the lightning pains of tabes is often a difficult matter. Among the most reliable drugs are phenacetin, antipyrin, antifebrin, and aspirin; sometimes success is obtained by one and sometimes by another, and occasionally a combination is very effective. They may be given conveniently in cachets, and, when the attack is very severe, Tourette recom-

mends 10 minims of tincture of opium after each cachet, provided that not more than 30 or 40 minims are given in 24 hours.

The use of aluminium chloride in diminishing the frequency of the attacks has already been mentioned.

Injectations of morphia will, of course, give relief, but, on account of the danger of the patient acquiring a craving for this drug, they should only be resorted to in exceptional cases.

The various crises must be treated by similar methods. In the gastric crises the difficulty of retaining drugs in the stomach often makes it necessary to give relief by hypodermic injections or by rectal suppositories.

Treatment of bladder troubles.—When it is necessary to draw off the urine the utmost care must be taken to use an aseptic catheter. When cystitis has occurred, urotropine and borax may be given, and, if necessary, the bladder must be washed out.

Treatment of ataxy.—To many patients the inability to walk straight is their most distressing symptom, and the methods introduced by Frenkel for correcting this difficulty by means of systematic exercises have proved to be of decided value.

Incoordination is the result of degeneration of nerve fibres from the muscles, joints, and ligaments, whereby the sense of position of the limbs is greatly diminished, often so much so that when the eyes are closed the patient is unable to tell at what angle his arms or legs are flexed.

The object of Frenkel's exercises is to improve the sensibility of the remaining fibres so that their increased function will fill the gap left by those that are degenerated.

In an average person the functions of the special senses are seldom, if ever, educated to the highest possible pitch—a fact which is clearly shown by the delicate sensibility for touch and

sound which may be acquired by the blind. In tabes, unless the degeneration is exceptionally extensive, there is a considerable margin left for the possibility of improving the muscle-sense, and by taking advantage of this possibility the incoordination can to a great extent be corrected.

To obtain success, the exercises must be performed slowly and accurately, taking great care to avoid fatigue, which, as stated above, is so harmful. It is necessary to insist upon this point, because the efforts made to move the limbs slowly and accurately are a great strain on the mind as well as on the body.

Various exercises can be devised without much trouble and expense. The patient may be taught to regain command over his legs by lying on a couch and raising the foot from square to square of a wooden frame so that the big toe is brought to rest smoothly at each movement, and moving the foot backwards and forwards so that the heel is placed accurately on various marked spots. To this may be added exercises in walking accurately to patterns on the floor, and, later on, walking sideways and other more difficult feats may be attempted.

The hands may be trained by means of draught and lialma boards, or some similar objects, by which accuracy and attention are brought to bear on the movements.

Schwab and Allison* have shown that in tabes the foot tends to assume a faulty position of eversion, and that by correcting this by suitable boots a mechanical advantage is obtained which further assists to diminish the incoordination.

* *Journ. of Amer. Med. Assoc.*, Dec. 16, 1905.

CHAPTER XVIII

FRIEDREICH'S DISEASE (HEREDITARY ATAXY)* AND HEREDITARY SPASTIC PARAPLEGIA

Etiology.—Although Friedreich's disease is known also as hereditary ataxy, the symptoms are seldom transmitted directly from one generation to another. On the other hand, the disease is often found in several members of a family.

Both sexes are liable to suffer. The first symptoms may be noticed somewhere about the age of puberty, but they often begin at an earlier date. Occasionally, acute infective diseases appear to have been the starting-point, but the degeneration can seldom, if ever, be definitely traced to any obvious cause, nor does it in any way appear to depend on syphilitic taint.

Pathology.—The main lesions comprise degeneration and sclerosis of the posterior, lateral, and direct cerebellar tracts of the cord, and sometimes also of the antero-lateral tracts. The primary change is probably a degeneration of the nerve fibres, to which the overgrowth of connective tissue is secondary.

* In some families, certain structures show a special disposition to atrophy prematurely. The reason for this is obscure, and can at present best be explained by assuming an inherent lack of vitality in the tissues, rather than the presence of an accidental toxæmia, for were the degeneration due to the latter it would not, probably, so frequently show itself in several members of a family. Premature degenerations of this kind occur in the nervous and muscular systems as well as in other organs of the body, and are included in the group to which the title of "abiotrophy" has been applied by Sir William Gowers.

Symptoms.—When the parents are observant, as they are likely to be if one child after another is struck down, a change in the shape of the foot is often the first warning that anything is wrong. In others, the clumsy movements of the legs or arms are the earliest signs. This clumsiness is due to the ataxy resulting from degeneration of the



Fig. 55.—Pes cavus, from a case of Friedreich's disease.

posterior and cerebellar tracts; it resembles that of tabes dorsalis, and is worse when the eyes are closed. The legs are generally first affected, and the child stumbles and falls about; later on, the disorder extends to the trunk and arms.

Weakness.—The pyramidal tracts are affected as well as the posterior columns; hence some weakness accompanies the ataxy, and the main symptoms in the legs are those of ataxic paraplegia.

Reflexes.—The continuity of the arcs for the tendon reflexes are usually broken at an early period of the disease, so that the knee-jerks are almost invariably lost.

The tendo-Achillis jerk and the deep reflexes of the upper limb likewise cannot be obtained.

When the plantar reflex is preserved it is often “extensor” owing to the degeneration of the crossed pyramidal tracts.

Deformities.—The shape of the feet alters, and assumes that known as “pes cavus,” in which the arch of the foot is exaggerated and the toes become deformed as “hammer toes” owing to the contraction of the tendons, the extensor proprius hallucis often standing out very prominently (Fig. 55).

A lateral curvature of the spine often develops.

The eyes.—There is usually a slow lateral nystagmus to be observed when the eyes are moved to one or other side. There is no Argyll-Robertson pupil, and optic atrophy has only rarely been observed.

Nodding movements of the head and a slurring, stuttering speech are both common.

Sensation.—In spite of the extent to which the posterior columns are sclerosed, there is seldom any appreciable loss of sensation, at any rate until the late stages of the disease. This shows how widespread are the paths through which sensory impulses are conducted up the cord.

Diagnosis.—A well-marked case can give rise to little difficulty, as the age at which it begins and the characteristic symptoms are very distinctive. The conditions most likely to cause confusion are juvenile tabes and disseminated sclerosis. Tabes acquired in later life can usually be excluded by the history.

The main points in which Friedreich's disease differs from tabes are set out in the following table.

	FRIEDREICH'S DISEASE.	LOCOMOTOR ATAXY.
AGE OF ONSET ...	About or before puberty	Later, except in rare cases of juvenile tabes.
PAINS ...	Absent ...	Characteristic light- ning pains.
ARGYLL-ROBERTSON PUPIL	Absent ...	Frequent.
OPTIC ATROPHY ...	Absent (almost always)	Frequent.
NYSTAGMUS ...	Present ...	Absent.
LOSS OF POWER ...	Present ...	Not common (unless quite local) till late stages of disease.

Disseminated sclerosis.—Disseminated sclerosis is in some cases differentiated with difficulty, since many of the symptoms are common to both affections; but the slow, unvarying progress of Friedreich's disease often contrasts with the variations met with in disseminated sclerosis, especially during the early stages. The coarser and more irregular tremor, the ocular palsies, and pallor of the optic disc all help to distinguish disseminated sclerosis.

Hereditary cerebellar ataxy.—Hereditary cerebellar ataxy of Marie very closely resembles Friedreich's disease; indeed it is probably impossible to make a definite diagnosis in every case.

The main points which generally help to differentiate hereditary cerebellar ataxy are the later stage of onset and the preservation or increase of tendon reflexes, together with the absence of spinal curvature and of deformities of the feet.

Variations from type.—It is plain that with such a wide distribution of degeneration considerable deviation must be occasionally expected from the type originally described by Friedreich. Where the columns degenerate unequally it must sometimes happen that the symptoms due to an

advanced condition of one predominate over those due to a slighter lesion of another tract, and this especially occurs when the lateral columns degenerate before the posterior and cerebellar. In such cases, spasticity and increased reflexes are the more prominent features. These cases generally complain first of deformity of the feet, and, from the youthful age of the patient and the history of the onset, the diagnosis of Friedreich's disease naturally suggests itself. Various other signs, *e.g.*, a certain degree of ataxia, nystagmus, and trophic ulcers of the feet, may lend confirmation to the diagnosis, though the last-mentioned symptoms are not usual in Friedreich's disease. The chief difficulty met with is in the condition of the knee-jerks, which are exaggerated. This, according to most authorities, is absolutely against Friedreich's disease.

Thus we may be faced with a set of symptoms which closely resemble those of Friedreich's disease, but yet differ markedly from the latter in the character of the knee-jerks, the absence of which authors regard as such an important sign. Such cases may be considered as "atypical cases of Friedreich's disease," yet, considering the frequency with which they occur, it is more satisfactory to class them separately as cases of **hereditary spastic paraplegia.**

The term hereditary is somewhat unsatisfactory here as in hereditary ataxy, since this condition is likewise familial rather than inherited, but it is best to retain it since the term hereditary ataxy remains in use.

We have, then, in hereditary spastic paraplegia a disease which shows itself clinically by symptoms which appear chiefly to depend upon a premature degeneration of the lateral tracts, to which may be superadded symptoms due to some degeneration of the posterior columns and occasionally of other parts of the nervous system.

There is a close relationship between it and Friedreich's disease, for the degeneration in both depends on some inherent lack of vitality; but it can be distinguished from the typical cases of Friedreich's disease by the predominance of the spastic symptoms, though occasionally, as might be expected, the two conditions practically merge into one another.

Prognosis.—The tendency of these spinal examples of abiotrophy is to progress slowly, but occasionally the cases seem to be stationary for years.

The author's impression is that the progress of disease in the hereditary spastic paraplegia type is slower and more likely to be arrested than in the Friedreich type.

Treatment.—No drugs are known to have any definite influence in checking the progress of the degeneration, but much can be done by maintaining the general nutrition by such drugs as cod-liver oil and iron, and by attending to the general health in every way. Under healthy conditions it is rational to assume that the progress of the disease is likely to be slower, and in some instances it may possibly be permanently arrested.

Locally, the condition of the feet must be considered, and, as already mentioned, in cases of hereditary spastic paraplegia, where there is often, for a time, no ataxy, the deformity of the foot is the patient's chief complaint. Here it is that the hammer-toes are so troublesome, for they are distorted in such a way that they are always rubbing and pressing against the boot and are apt to become covered with very painful corns. While a suitable boot is very helpful in relieving this tendency, it is often necessary to devise some additional simple mechanical appliance. When the foot is flat on the ground it will be observed that the toes are almost straight, and in that position would run very slight risk of being chafed by the

boot, but the moment the foot is raised, up go the toes. The object of the mechanism illustrated in Fig. 56 is to keep the toes as far as possible

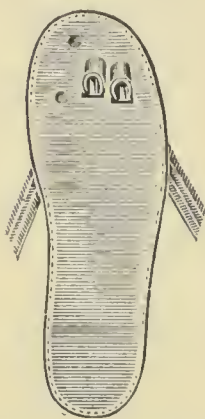


Fig. 56. — Device for dealing with hammer-toes.

in the position which they are still able to assume when the naked foot is placed flat upon the ground. A sole of firm leather is made to fit the under surface of the foot, and opposite the toes is a tape which passes through properly - constructed holes in the leather sole, and thus enables the toes to be laced down. The tape, of course, can be changed as often as necessary. It is simple and inexpensive, and patients state that they find great benefit from it.

As a rule, the progressive nature of the disease, the number of tendons and the amount of fascia involved in the deformities, as well as the likelihood of difficulty in getting the parts to heal well, make these cases unfavourable for operation.

CHAPTER XIX

SUBACUTE COMBINED DEGENERATION

IN this variety of combined sclerosis the degeneration runs a more rapid course, and is more particularly associated with severe anæmias or other toxic diseases to which allusion has been made under the heading of Ataxic Paraplegia.

Symptoms.—The disease usually occurs in middle-aged people, and it has been shown by Risien Russell, Collier, and Batten that many of these cases pursue a fairly definite course which can be divided into stages, but in others no such sharp distinctions can be made.

In the first and longest stage the symptoms are somewhat similar to those described under the heading of Ataxic Paraplegia, viz., weakness and rigidity, with increased reflexes, and a variable amount of ataxy. With these signs there are generally pronounced sensations of tingling and numbness and pains of the lower limbs, to which similar symptoms in the arms may be added.

In the next stage there is added a loss of muscle and cutaneous sensibility, the latter beginning in the legs and extending upwards on to the trunk. The spasticity during this period also becomes more marked, and is accompanied by increased tendon reflexes, extensor response, and the other signs of an upper motor neuron lesion.

After some weeks or months there comes the third stage, in which the spasticity is rapidly superseded by flaccidity, with loss of tendon

reflexes, wasting of muscles, and loss of control over the sphincters. The extensor plantar response remains. Edema of the legs, bed-sores, and cystitis may now occur, fever is present, and the patient loses strength and usually dies within a few weeks.

Pathology.—The cord shows degeneration chiefly of the lateral and posterior columns, secondary to a more localised and diffused transverse lesion, which is generally most marked in the thoracic region of the cord.

Diagnosis.—The difficulties in diagnosis will vary according to the stages at which the disease is seen.

When the signs are those of spastic weakness and ataxy the likelihood of *disseminated sclerosis* being present has to be considered. The main points in which disseminated sclerosis differs are in the earlier age of onset, the variableness of symptoms, the tendency to optic atrophy and cranial nerve palsies, and the presence of nystagmus.

As the disease progresses the degeneration may be referred to the results of a previous transverse *myelitis* or accounted for by the presence of a chronic myelitis.

When the knee-jerks are lost and there is flaccid paralysis, peripheral neuritis has to be thought of, but the anæsthesia of the trunk, the incontinence of urine, and an extensor plantar response are against it, as also is the history of the disease.

The loss of power, wasting and electrical changes in the muscles, together with absence of the Argyll-Robertson pupil, will enable *tubes* to be excluded.

Sometimes the signs may suggest the presence of a *tumour*, but the pains are unlike those due to pressure on roots, and the paralysis and sensory changes do not show the signs of being due to pressure on one half of the cord more than the other, as often happens in the case of tumours.

In the earlier stages subacute combined degeneration may be diagnosed as *hysteria*. In doubtful cases the most reliable sign of organic disease is to be found in the extensor response of the plantar reflexes. If these are obtained, the organic nature of the case is clear; and where there is any uncertainty, they should be repeatedly examined, since they do not always respond in the same manner.

Prognosis and Treatment.—Hitherto the cases recorded seem all to have ended in death, nor has their course been materially modified by any drugs. Treatment must be directed towards modifying the general health, which is always so bad, and towards improving the condition of anæmia which is so generally present.

Differential
Examination & clinical
myelitis
Cerebral haematomata
Tubercles
Leucæmia
Hysteria

CHAPTER XX

DISSEMINATED SCLEROSIS

Etiology.—Very little is definitely known about the origin of disseminated sclerosis.

While there is no exact age limit, the symptoms begin in most cases between the years of 15 and 35. Children occasionally suffer, but elderly people are very rarely attacked.

The number of cases occurring in the different sexes is about equal.

Pathology.—The disease, as its name implies, is characterised by the formation of islets of connective tissue which are scattered throughout the length of the central nervous system and also to some extent in the substance of the peripheral nerves, more especially in those of cranial origin. The disease in classification therefore belongs both to the brain and the cord. The patches vary greatly in size. Some are so small as to be scarcely discernible by the naked eye, while others are as large as peas. The white matter of the brain and cord is almost exclusively affected, it being quite rare to meet with symptoms attributable to alterations in the grey substance.

The changes appear to begin in the neuroglia rather than in the nerve fibres, though on this point there is some difference of opinion. In any case the axis cylinders are the last structures to degenerate, which accounts for the fact that there is seldom any secondary degeneration to be detected either above or below the sclerosed site.



PLATE VII.—Instantaneous Photograph showing Tremors on drinking a Glass of Water in a Case of Disseminated Sclerosis.

ye Symptoms . Nystagmus. best seen on
voluntary movement of the eyes - Other
ocular signs such as impairment of
vision & optic atrophy may be present -
transitory squinting or diplopia may
occur

Before, however, the patches are permanently formed there is frequently a preliminary period during which the lesions are subject to variation. These early changes may be due to vascular disturbances with possibly some passing œdema, and their variations correspond to the remissions and exacerbations which clinically are so characteristic of the disease.

The principal suggestions which have been put forward to account for the origin of the patches are:

(1) That they are due to the presence of toxins.

(2) That they are the result of some congenital abnormality of the nerve tissue.

In favour of the first view is the evidence that the symptoms sometimes appear to arise after the infections of specific fevers. Against this, however, is the fact that the white matter is almost exclusively affected, and in many instances where a specific fever appears to have brought on the symptoms it has doubtless only aggravated them, or perhaps precipitated their occurrence. Congenital changes, probably beginning with a proliferation of neuroglial tissue after the manner of that which takes place in syringo-myelia, are on the whole more likely.

Symptoms.—In the form which has generally been considered characteristic of the disease the symptoms are mainly weakness, spasticity, and ataxy of the lower limbs, together with alterations in speech, tremors, nystagmus, and optic atrophy. This combination of signs makes up a very striking picture.

The patient walks with some degree of difficulty: the feet shuffle on the ground owing to the rigidity which impairs the freedom of the arthritic movements, while there is also an unsteadiness of gait owing to changes in the posterior and cerebellar tracts.

When the patient is seated, but little is noticed, yet any movement of the arm or hand is accompanied by a coarse tremor, which increases in amplitude as the movement is continued. Thus, if the patient be asked to carry a glass of water to his lips, the movements become more and more exaggerated, until the contents of the glass are dashed to the ground (Plate VII.). In the same way, if he be asked to write, the pen is drawn spasmodically across the paper, though, of course, in the earlier stages, all grades of impairment of writing are encountered. In those engaged in clerical work this symptom is sometimes the first noticed. This tremor, often known as "volitional" or "in-

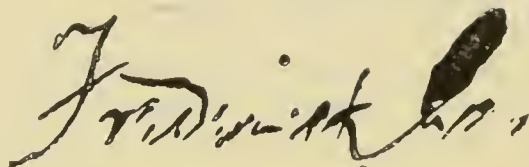


Fig. 57.—Specimen of writing in a case of disseminated sclerosis.

tentional" tremor, is by no means necessarily confined to the arms. Sometimes there are rhythmical movements of the head, which, however, cease when the muscles of the neck are supported on a pillow.

The tremor of disseminated sclerosis, then, is characterised by—

- (1) Its occurrence during voluntary movements.
- (2) Its cessation during rest.
- (3) The coarseness of its vibrations, which increase in amplitude with the continuation of the movement.

The power of the legs (and probably of the arms) will be found to be considerably less than normal

The knee-jerks are increased, ankle-clonus is often obtained, and the plantar reflex shows the

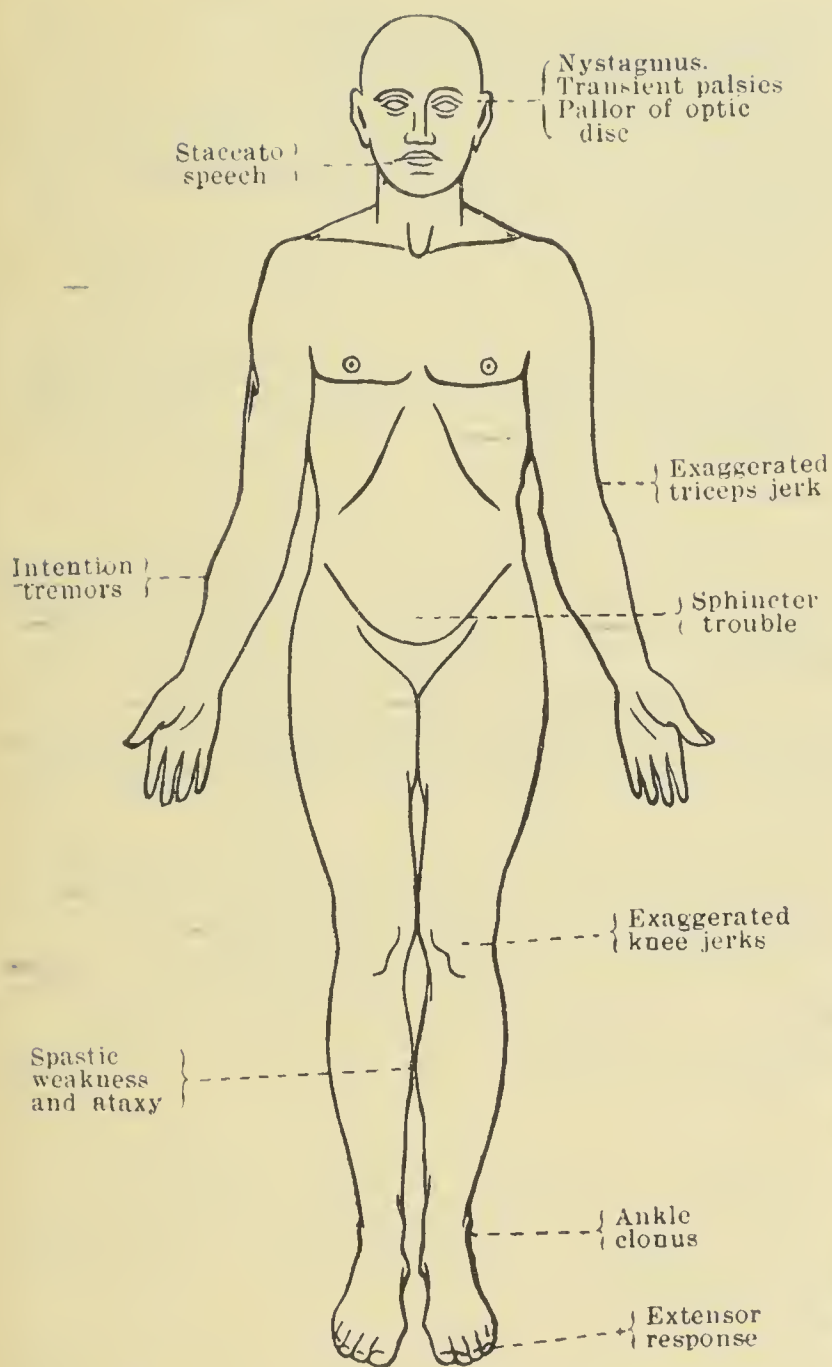


Fig. 58.—Summary of the principal symptoms occurring in the course of disseminated sclerosis.

extensor response; there may also be some tendency to clubbing of the feet.

In the upper limb also the deep reflexes are generally found to be exaggerated, and a brisk jaw-jerk will in all probability be obtained, but the superficial reflexes of the abdomen are frequently lost.

As regards the ocular signs, nystagmus is observed when the patient attempts to look to one or the other side, and there may be some weakness (usually transient) of one or more muscles giving rise to double vision. The pupils usually react normally to light and during accommodation. Changes in the optic nerves occur in a large proportion of cases of disseminated sclerosis. The alteration most commonly seen is that of undue pallor of the optic disc, indicating a certain degree of atrophy. In a very small percentage of cases, signs of antecedent neuritis have been noted. The atrophy is often confined to the temporal half of the disc and it frequently affects only one eye. Only exceptionally, therefore, is total blindness likely to occur. The fields of vision may show restricted perception of colours before the sensation of ordinary light is demonstrably impaired.

The speech is known as "scanning" or "staccato," from the pronounced way in which every syllable is jerked out.

There is generally some impairment of sphincter control at some time during the course of the disease.

Mental instability is often apparent, as is shown by a tendency to laugh and cry when there is but slight emotional stimulus; occasionally epileptiform fits occur.

Such are the main points to be observed in a so-called typical case, but it by no means follows that the signs always appear in this pronounced form; indeed the majority of patients when first seen do not present a picture anything like this,

and may go on for years with vague and transient symptoms, the cause of which is often attributed to hysteria, unless careful examinations are repeatedly made. It therefore happens that these so-called atypical cases are of the greatest importance because (1) numerically they are greater than those in which signs are from the first permanent and pronounced, (2) they are so apt to be diagnosed as functional, and (3) any advantage that can be obtained from treatment is likely to be far greater when the changes in the nervous tissue are capable of variation than when they have become permanent.

The patient, very commonly a girl, and usually somewhat above the age of puberty, may complain of peculiar sensations of numbness which come and go in a mysterious manner, and for which no obvious cause can be found, although they sometimes tend to be more severe after fatigue or at the time of the menstrual period. Examination during such an attack may or may not reveal a patch of anæsthesia which does not seem to correspond to any definite anatomical arrangement, and then perhaps everything passes off for a time, and all is forgotten until, later on, one of the legs drags, or all power suddenly goes out of the arm, again to return or vary from time to time. It may be (and this is very significant) that there is a temporary complaint of double vision, and there is very often transient difficulty in exercising full control over the sphincters.

In all such cases the greatest care must be taken before organic disease can be excluded, and where the history is suspicious and the results of examination negative it is often wise, if possible, to postpone giving a definite opinion, for signs which are absent one day may often be elicited with ease on another.

Pallor of an optic nerve, nystagmus, squint, slight incoordination of the arms, and an extensor

response of the plantar reflexes are among the most important things to note, and the establishment of one sign which is of definitely organic origin is as useful in a difficult case as is the discovery of a single tubercle bacillus in a case of suspected pulmonary disease.

In others, although the symptoms are obviously of organic origin, they may for a time be so restricted as to give rise to doubt regarding their nature, as, for example, when a spastic or an ataxic paraplegia is the only condition present.

In yet others the patches of sclerosis, though widely distributed, may not be in the positions to produce the typical signs of the disease, and thus arises another set of cases not conforming to type.

Diagnosis.—It will be evident that the diagnosis of disseminated sclerosis may be very easy or the reverse, and further, that the difficulties will vary during the different phases of the disease.

1. During the early stages of its development the tendency is to mistake it for hysteria. The chief points to be noted in making the diagnosis have already been mentioned.

2. When the patches are situated exclusively in the course of the fibres of the lateral tracts, primary lateral sclerosis will be thought of, and, in the absence of any further signs, time alone may determine the diagnosis; but it must be remembered that primary lateral sclerosis is rare, and that many cases which appear at first to be of this nature eventually develop signs of the disseminated type.

Aid may often be obtained from the variability of symptoms, for primary lateral sclerosis is a disease of gradual but steady progress, whereas remissions are very characteristic of disseminated sclerosis.

When there are patches in the posterior in addition to the lateral columns, and no symptoms

point to sclerosis elsewhere, the diagnosis for a time may be ataxic paraplegia, but this may require correction on the appearance of other signs. It is, therefore, wise, when making the diagnosis of ataxic paraplegia, to keep in mind the further developments which so many of those cases undergo.

3. When the signs are well developed, yet are not those which characterise the recognised types of the disease, there may be hesitation in distinguishing the condition from Friedreich's ataxy, paralysis agitans, cerebellar disease, and, in children, from cerebral diplegia. General paralysis of the insane sometimes shows symptoms which

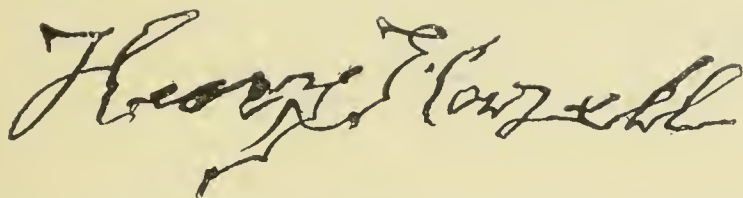


Fig. 59.—Specimen of writing from a case of chronic poisoning by mercury.

resemble those of disseminated sclerosis, but the inequality of the pupils and the loss of the light reflexes, together with the mental changes present, do not usually leave the diagnosis in doubt.

Paralysis agitans can seldom give rise to real difficulty, for the tremor is finer, occurs during rest, and is diminished rather than otherwise on movement, while rigidity is seldom absent.

Confusion with cerebellar disease is only likely to arise in very chronic cases. In the more acute, the optic neuritis, headache, and vomiting are distinguishing features. Cases of cerebellar ataxia of childhood may sometimes resemble disseminated sclerosis, as also may some of the less typical instances of cerebral diplegia. A tremor which closely resembles that of disseminated sclerosis often occurs as a result of chronic mercurial poison-

ing, but the history of the case, together with the absence of other signs of the former disease, makes the diagnosis easy.

Prognosis and Treatment.—The outlook for patients with disseminated sclerosis is not hopeful, since experience has shown that, despite the transient nature of certain early symptoms, a temporary improvement is the most that can be expected. Still, if the cases come under treatment early, it is doubtless possible frequently to retard the course of the disease by insisting on the avoidance of fatigue during the stages of remission, and on complete rest during those of exacerbation.

At the same time the general nutrition should be carefully maintained by a diet containing an abundance of milk. Tonics, especially those containing iron, are useful; arsenic also has gained some reputation in this disorder, but no drug is known to have any specific effect upon the lesions. Massage, carefully regulated, is of use during certain phases, and in the more chronic conditions selected exercises may be tried with a view of improving the accuracy of the movements.

Hysteria

Primary Lat. Sclerosis

Ataxic Paraplegia

Mercurial poisoning

Cerebellar Disease

Paralytic Ergasia

Friedreich's ataxia

Id. G. R. I.

CHAPTER XXI

MYELITIS

Etiology.—The nature of the lesions which are included under the clinical term “myelitis” varies. In some cases there is undoubtedly a primary inflammation brought about by the direct invasion of bacilli or by the effects of their toxins; in others the underlying condition is one of thrombosis, to which the changes in the cord are secondary.

It will, therefore, be evident that the acute infective diseases and syphilitic endarteritis are the most common causes. Cold, injury, and debility from any cause may, by lowering the resistance, dispose to an attack.

The existence of a “chronic myelitis,” in the sense of a persisting primary inflammatory process, is doubtful; most of such cases are probably of syphilitic origin.

The most vulnerable part of the cord appears to be the lower dorsal region, and it is there that the lesion most frequently occurs.

Pathology.—The substance of the cord is softened, and in some cases the different structures are indistinguishable from one another. Hæmorrhages, engorgement of vessels, exudation, and cell degeneration are all present in varying degree.

Symptoms.—The symptoms vary with the level and the extent of the lesion.

1. Transverse myelitis in the dorsal region.—When the onset is acute, and the patient has

hitherto been well, the first signs are generally a feeling of increasing heaviness in the limbs, with perhaps some difficulty in passing urine. The numbness and weakness rapidly increase, and, in a severe case, the lower limbs may become within a short time both senseless and motionless. Examination shows loss of power below the level of the lesion, and, assuming the attack to be severe, there will be absence of all movements of the feet, knees, or hips, the limbs lying flaccid and helpless. At this early stage the tendon reflexes may be temporarily inhibited through irritation of the pyramidal fibres at the level of the disease, but they soon become increased, as in other upper motor neuron lesions. At the same time that impulses are prevented from flowing down the motor tracts, the transverse lesion also prevents stimuli from passing up the sensory columns; hence there is loss of sensation of every kind below the level of the lesion, but at that level, and just above it, there is often a zone of hyperæsthesia, due to irritation of the sensory roots as they pass out from those segments of the cord. This zone of hyperæsthesia and the upper limit of the anæsthesia are valuable guides to the position of the lesion.

Total loss of sensibility is often accompanied by the formation of bedsores or crops of herpes, both of which are due to those disturbances in nutrition generally described as "trophic." No impulses from the bladder and rectum can reach the brain, nor can any pass downwards, with the result that control over the sphincters is lost. The bladder either discharges its contents reflexly at irregular intervals, or, as more frequently happens, it becomes distended until the incontinence of overflow ensues.

If the patient lives, the acuter symptoms pass off, and the permanent effects of degeneration subsequent to the transverse lesion become apparent



PLATE VIII.—Instantaneous Photograph showing Gait in Paraplegia following Myelitis.

(Plate VIII.). The fibres of the crossed pyramidal tracts degenerate downwards, with all the accompanying attributes of lateral sclerosis. The legs become *rigid*, with or without some return of power according to the intensity of the initial lesion, and the rigidity is succeeded by *contractures*, in which the strength of the flexors and adductors overpowers that of the extensors and abductors. The *tendon reflexes* are very exaggerated, *clonus* appears, and the *plantar reflex* gives a typical extensor response. There is no wasting, and no electrical changes are present. Whether control over the sphincters is partially or entirely regained depends upon the amount of communication that is re-established after the acute symptoms have subsided.

Such are the main points to be observed in a transverse myelitis of the lower dorsal region.

2. In the *lumbar region* a transverse lesion destroys many motor cells of the anterior horns, from which fibres go to supply the muscles of the legs, and hence there is a considerable amount of wasting. At the same time, the centres for the bladder and the knee-jerks are liable to destruction; consequently the superficial and deep reflexes disappear, and the bladder becomes a flaccid bag from which the urine continually runs, *i.e.*, true incontinence, as opposed to the false incontinence of the distended bladder. As in transverse myelitis of the dorsal region, there will be anæsthesia below the level of the lesion.

3. In the *cervical region* there will be paralysis and anæsthesia below the level of the lesion, as in the case of the dorsal region. In addition, there is the possible presence of hyperpyrexia and of inequality of the pupils (due to interference with the cervical sympathetic nerves). If the lesion chances to be at the level of the cervical enlargement, there will be wasting of the muscles of the arm from destruction of their motor cells, just as

the muscles of the leg waste when the lumbar enlargement is affected. If the motor cells of the phrenic nerves at the level of the third, fourth, and fifth cervical roots are destroyed, death takes place from failure of respiration.

Acute ascending myelitis, in which the inflammatory process commences in the lower parts of the cord and extends upwards, sometimes occurs. The chief symptoms are rapidly ascending paralysis with loss of sensation, loss of control over the sphincters, and the appearance of bedsores.

The whole diameter of the cord is acutely inflamed, and its structure destroyed. According to Buzzard, the infection probably takes place through the lymphatic system. *Disseminated acute myelitis*, in which the inflamed areas are scattered throughout the cord, is sometimes found.

Diagnosis.—In making a diagnosis it is first necessary to be certain that the lesion is in the cord, and the methods of ascertaining this point are described under the heading of Paraplegia (Chapter XIII.). When this is established it is necessary to consider the nature of the lesion. The acute onset suggests an inflammatory origin. Hæmorrhage into the substance of the cord, besides being very rare, has a more sudden onset, and would be unlikely to assume a transverse distribution, though bleeding into a cavity of syringomyelia might in certain circumstances give rise to difficulty.

The back must always be carefully examined to see if the symptoms may possibly be due to pressure.

Meningitis may simulate myelitis, but in the former there are usually radiating pains due to irritation of the nerve roots; the two conditions may, however, co-exist, as often happens in syphilitic disease of the cord.

An acute myelitis which ascends the cord will closely resemble a case of Landry's paralysis, but

in the latter there is usually no loss of sensation, no loss of control over the sphincters, no wasting, and no electrical changes in the muscles.

When the patient is seen for the first time after the acute stage has passed, the resulting degeneration will be in evidence.

The diagnosis will then have to be made from ataxic paraplegia, subacute combined degeneration, and disseminated sclerosis. The decision must depend largely on the history of the onset and the course of the disease, since the physical signs will often be compatible with diseases of different origin.

Prognosis.—Myelitis of the cervical region of the cord always threatens to destroy life by its proximity to the nuclear origin of the phrenic nerves and to the structures of the medulla. When the disease is situated in the dorsal and lumbar region there is less immediate danger, but if the structures of the cord are permanently destroyed the exhaustion accompanying bedsores and cystitis gradually carries off the patient. Where partial recovery takes place the patient may, with care, live very many years.

At the beginning of a case it is impossible to predict with any certainty how much power will be recovered. Some patients recover in an extraordinary way, but the majority, after reaching a certain point, make no more progress, and for the rest of their lives suffer from various degrees of spastic paraplegia, with or without some difficulty in exercising efficient control over the sphincters.

Treatment.—The patient should lie on his side or stomach, to avoid congestion of the diseased part. Attention must be paid to the state of the bladder, and the urine should be drawn off whenever necessary, during which operation the strictest aseptic precautions must be taken to prevent cystitis. He should lie on a water-bed, and the greatest care must be exercised to prevent

the occurrence of bedsores. The bowels should be kept freely open. The best medicinal treatment is mercury by inunction. Many of these cases are probably due to thrombosis produced by syphilitic disease of the vessels, and, even in the cases that are not of such origin, mercury often appears to exercise a beneficial influence. Counter-irritation is sometimes applied to the spine. When the acute symptoms subside strychnine is useful, but it should not be given if much rigidity is present. Massage, good food, and general tonic treatment are indicated at this stage, and every effort must be made to prevent the occurrence of contractures.

DIVER'S PARALYSIS (CAISSON DISEASE)

Symptoms analogous in many ways to those of acute myelitis are apt to arise when a sudden change is made from a very high to a very low atmospheric pressure such as may take place in the case of divers. The motor symptoms are usually those of paraplegia, which may either gradually pass off or be followed by spastic paralysis such as occurs after myelitis; but the disease may injure all the structures of the cord and cause anæsthesia and loss of control over the sphincters.

The injury is supposed to be caused by the rapid escape from the tissues of air which has been absorbed under a high pressure, and in order to avoid this danger the transition from high to low pressure should be gradual.

CHAPTER XXII

LANDRY'S PARALYSIS

THIS disease, first described by Landry, is characterised by a paralysis which begins in the feet and rapidly extends upwards to the trunk, arms, and bulb; occasionally the parts may be invaded in the opposite order. Both sexes may be attacked, and most of the cases have occurred between the ages of 20 and 45.

Etiology.—There can be little doubt that the symptoms are the result of a poison affecting the functions of the spinal cord, and sometimes they have appeared to be the direct sequelæ of attacks of septicæmia, typhoid, influenza, and other specific fevers.

Pathology.—No definite changes in the cord can be seen with the naked eye. Microscopically, hyperæmia and swelling of the cells have been observed. Changes in the peripheral nerves have also been described.

Symptoms.—Weakness begins in the legs, usually in one a few hours before the other, and in a very short time there is complete flaccid paralysis. The loss of power spreads upwards to the muscles of the abdomen, thorax, arms, neck, and face.

The muscles are flaccid, but usually show no definite changes in their electrical reactions and little or no signs of wasting. The tendon reflexes are lost, but the power of the sphincters is generally retained. As soon as the paralysis spreads

into the cervical region the patient is in imminent danger of death from paralysis of the diaphragm, and if he escapes this danger another is frequently met with from failure of the muscles of deglutition, with consequent tendency for food to regurgitate through the nose and to find its way into the larynx. Sensation is but little, if at all, altered, and the mind remains clear. The temperature may be raised, but pyrexia is not a prominent feature. The spleen is generally enlarged.

Variations.—Inasmuch as the nature of the poison is probably not the same in all cases, and the anatomical changes vary, it is not surprising that all cases do not adhere rigidly to the one type. In some the paralysis appears first at the upper end of the body and spreads downwards. In others, especially those which last a long time, there may be signs of wasting of the muscles and changes in their electrical reactions, while in others again the action of the sphincters may be impaired.

Nevertheless, there is clinically a very definite set of cases characterised by a paralysis which ascends and which is unaccompanied by wasting or sensory disturbances.

The most practical view is to suppose that certain poisons, under certain conditions, lower the functions of the cells of the anterior horns (and perhaps other parts of the nervous system as well) sufficiently to produce a flaccid paralysis, but insufficiently to cause wasting and electrical changes; that here and there the intensity of the poison is sufficiently great to lead to such changes, and that gross anatomical lesions sometimes further intensify the symptoms. In this way we can understand the variableness of the symptoms in different cases.

Diagnosis.—The definite march of the paralysis, and the absence of wasting, of electrical changes, and of altered sensation generally, enable

the disease to be distinguished, though, from what has already been said, it will be plain that the less typical cases may occasion difficulty.

From peripheral neuritis Landry's paralysis differs in the extension of the paralysis to the trunk before the arms are affected, and also in the absence of wasting and of electrical changes, which are generally soon seen in peripheral neuritis.

In an ascending myelitis, where there is a definite inflammation spreading up the cord, there will be loss of control over the sphincters, and bedsores will soon occur.

An infantile paralysis (acute anterior poliomyelitis) in which the symptoms are widespread may be difficult to differentiate for a time, since both this and Landry's paralysis depend upon lesions of the motor cell of the anterior horns. Usually the more scattered paralysis of the poliomyelitis makes the diagnosis clear, but where there is a difficulty it may be necessary to wait until wasting and electrical changes appear in the muscles. When a patient recovers from Landry's disease the power returns more rapidly than in infantile paralysis.

Prognosis.—A patient suffering from Landry's disease is always in great danger until the acute symptoms have subsided and the risk of respiratory failure has passed.

In a large number of cases the disease ends fatally in a few days, while in some the symptoms subside and power gradually but steadily returns.

Treatment.—In the absence of any specific antidote to the poison, reliance has to be placed on maintaining the patient's strength, and for this purpose hypodermic injections of strychnine are probably the best. Ergot has been recommended.

The breathing of the patient should be carefully watched, and an attempt made to tide over any temporary difficulty by artificial respiration.

CHAPTER XXIII

SYRINGO-MYELIA

OVERGROWTH of neuroglia and the formation of cavities in the spinal cord are the chief characteristics of syringo-myelia.

Etiology.—The disease occurs in both sexes, and the symptoms are generally first noticed in the early years of adult life.

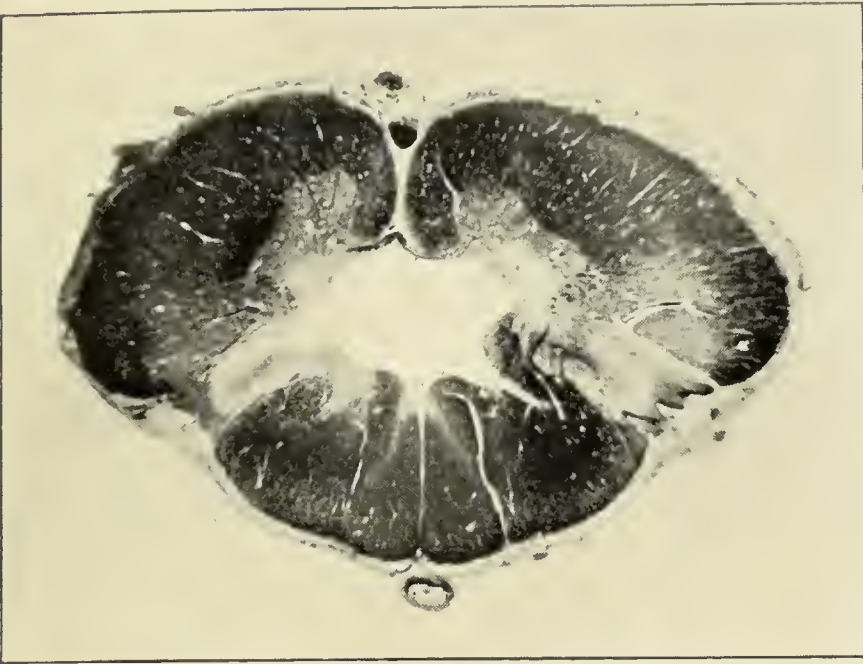
Injuries, inflammations, and vascular lesions occasionally seem to be the starting-point of the disease.

Pathology.—Different views are held concerning the origin of syringo-myelia. By some it is considered that there is a congenital developmental defect in the cord which results in distension of the central canal and proliferation of the neuroglial tissue on its boundaries; and in support of this hypothesis there are the cases in which the cavities have been found to be lined with epithelium similar to that of the central canal.

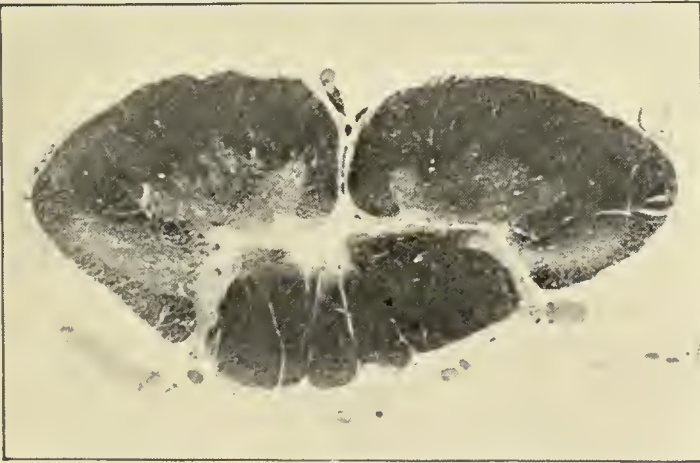
Others consider that proliferation of the neuroglial tissue is the primary process, and that the formation of cavities is secondary to degeneration. It has further been suggested ~~that the gliosis~~ is due to some irritant poison, but this at present is a matter of speculation. The cavities vary both in shape and position, thus accounting for the variations that are met with in the symptoms during life.

The cavities are generally situated in the grey

A



B



C



PLATE IX.—Photographs showing Cavities (A and B) and Gliomatosis (C) of the Spinal Cord in Syringo-myelia. (*Pierre Marie.*)

matter near the centre of the cord, but they often extend into the anterior horns, and sometimes into the posterior horns also. The walls of the cavity are generally formed by the proliferated neuroglial tissue (Plate IX.).

Symptoms.—The symptoms are due (1) to loss of cord substance, and (2) to pressure on surrounding parts by the proliferating neuroglia, which in some cases amounts to a definite tumour formation. The symptoms must, of course, differ with the position and size of the cavity and the degree of gliosis round it, but as the grey matter is especially liable to be affected there is a certain degree of uniformity in the main symptoms of any consecutive number of cases.

(1) The lesions of the grey matter interrupt impulses of pain and temperature, hence there is usually *loss of sensation to heat, cold, and pain*, while the tactile sensibility frequently remains unimpaired (Figs. 60 and 61).

(2) Injury of the motor cells of the anterior horns occurs from extension of the cavity or from pressure of gliomatous tissue, and is followed by a slow, progressive *wasting of muscles*.

(3) There are also *trophic lesions*, but the mechanism by which they are produced is uncertain.

The excavations are usually most extensive in the cervical region of the cord, so that the wasting of muscles is most prominent in the arms, while, at the same time, pressure on the lateral tracts often gives rise to some degree of weakness in the legs together with increase of knee-jerks, ankle-clonus, and extensor response of the plantar reflex.

Trophic disturbances are common, some of which probably arise in the first place from burns and injuries which have not been felt. Spinal curvatures also occur.

Painless whitlows, arthropathies resembling those of tabes, vasomotor disturbances of the skin

causing localised anemias or hyperæmias, excessive or deficient perspiration, bullous eruptions, local gangrene, and perforating ulcers, are all met

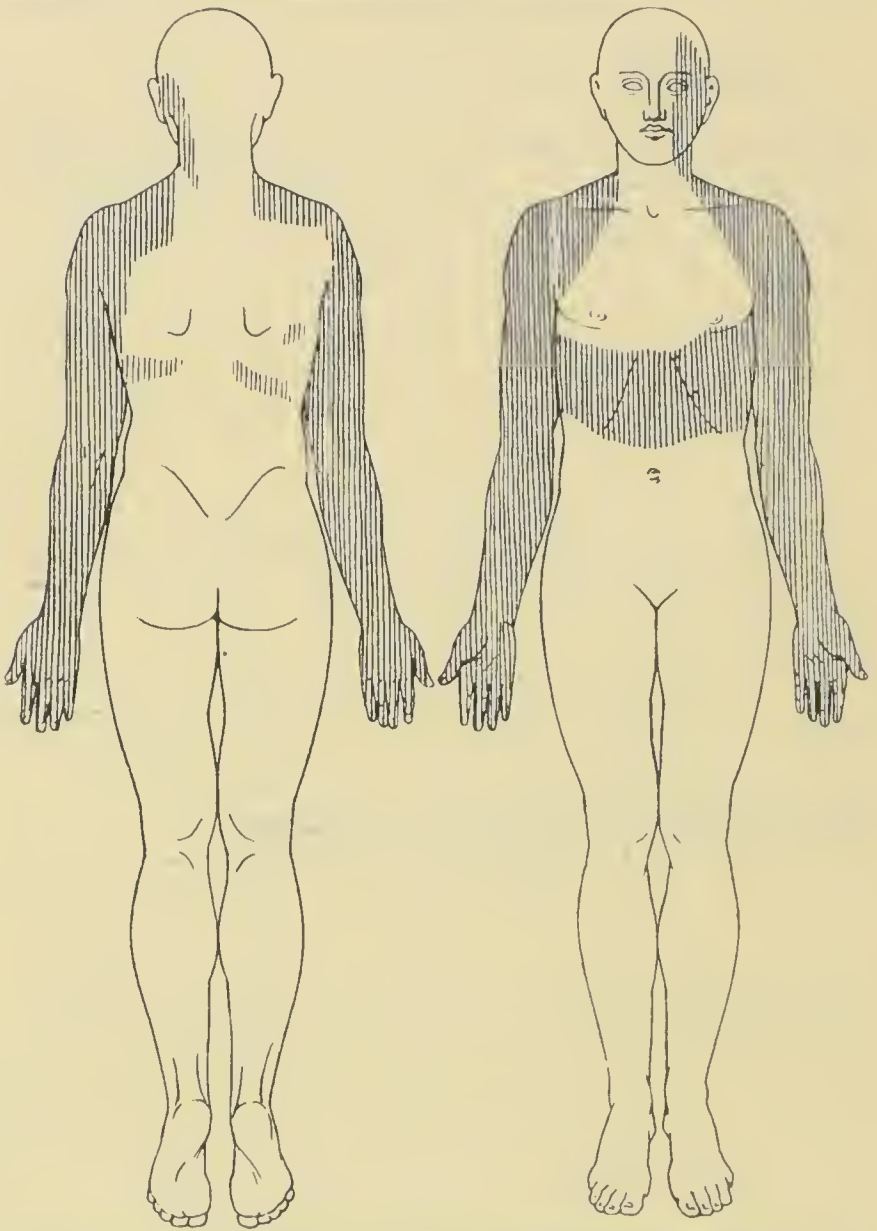


Fig. 60. — Diagram showing loss of sensation for heat and cold in a case of syringo-myelia.

with. The painless whitlows in conjunction with other nutritional disturbances of the hands were first described by Morvan, but it is generally con-

sidered that "Morvan's disease" is a variety of syringo-myelia.

In some cases the morbid processes spread up-

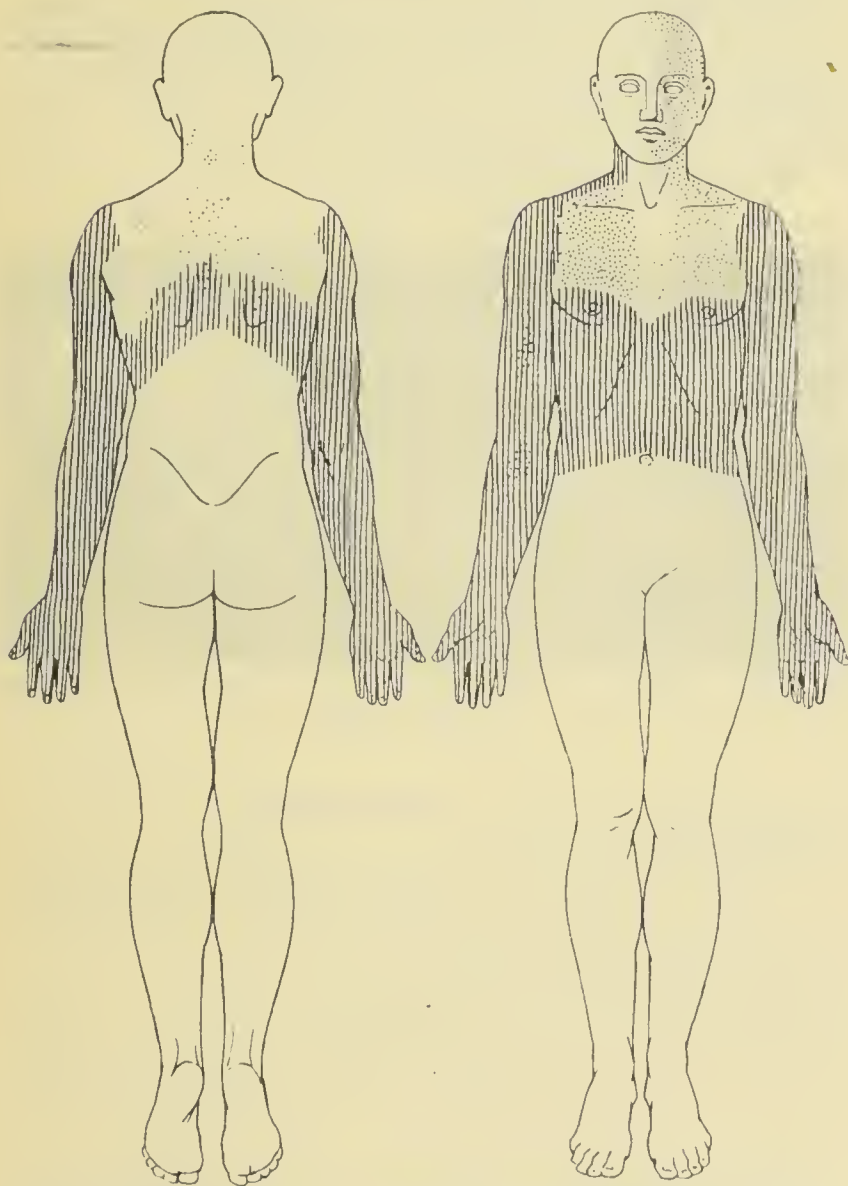


Fig. 61.—Diagram of the same case showing areas over which sensations of pain were lost or blunted.

wards to the medulla, and atrophy and weakness, sometimes one-sided, of the tongue, face, and palate may occur. Nystagnus is also occasionally seen.

If the cavity is in the lower part of the cord the symptoms may be mainly confined to the legs.

Diagnosis.—The wasting of the hands may lead to the diagnosis of *progressive muscular atrophy*, unless the sensations for pain and temperature are tested, when the real nature of the condition will be apparent.

Where sensory disturbances are present alone,



Fig. 62.—Trophic changes and deformities of fingers from a case of syringo-myelia.

~~care must be taken not to put them down to hysteria.~~

When the cavities are mainly in the posterior horns the symptoms may for a time simulate those of *locomotor ataxy*.

Prognosis.—The tendency is for the disease to progress slowly. Now and then there are exacerbations in the symptoms which sometimes appear to be due to hæmorrhages taking place into the cavity.

Treatment can only be directed towards keeping up the nutrition of the wasting muscles.

diff
Progressive muscular atrophy
locomotor ataxia
hysteria

CHAPTER XXIV

SPINA BIFIDA; HÆMATO-MYELIA

SPINA BIFIDA

SPINA BIFIDA arises from maldevelopment of the laminæ of the vertebræ, with which malformations of the cord and its membranes are frequently associated.

Varieties.—The following five varieties may, according to Bland-Sutton,* be recognised:

(1) Myelocoele, in which the central canal of the cord is open and from which the cerebro-spinal fluid continually drains away. This variety is more common in still-born children, and those who are born alive usually do not survive more than a few days.

(2) Syringo-myelocoele, in which the central canal is dilated and forms the cavity of the protruded sac. This form is exceedingly rare.

(3) Meningo-myelocoele, in which the membranes and the cord both protrude, but the cord, instead of being dilated, is usually flattened on the posterior wall of the cyst. This is the commonest form.

(4) Meningocoele.—In this variety there is a protrusion of the membranes only, the cord remaining normal.

(5) Masked spina bifida (spina bifida occulta), in which there is a deficiency of one or more of the vertebral arches which is not associated with any

* "Tumours, Innocent and Malignant," 4th Edition. Cassell and Co., Ltd., 1906.

abnormality or protrusion of the cord or membranes.

Spina bifida is apt to be associated with symptoms of hydrocephalus and syringo-myelia. It also gives rise to various forms of talipes and nutritional disturbances, such as perforating ulcer. Other deformities of developmental origin may also coexist.

Treatment.—In cases where it is possible to do so, excision of the sac is probably the best form of treatment. Many cases are best left untouched and protected by a suitable shield. In simple meningoceles good results are sometimes obtained by the injection of Morton's fluid, which causes the sac to contract. Morton's fluid consists of: Iodine, gr. x.; potassium iodide, gr. xxx.; and glycerine, ad ʒi. From mxxv. to ʒi. is the amount usually injected at a time.

HÆMORRHAGE INTO THE SPINAL CORD (HÆMATO-MYELIA)

Etiology.—Hæmorrhage into the substance of the cord may be associated with injuries to the spine, or it may occur into an area of the cord already diseased, *e.g.*, in myelitis, tumours, and syringo-myelia.

A primary spinal cord hæmorrhage analogous to that met with in the brain is very uncommon.

Symptoms.—The symptoms depend upon the position and the extent of the hæmorrhage. Sudden pain in the back and paralysis are usually first met with, and in transverse lesions there is loss of sphincter control, together with anæsthesia of the parts below the lesion.

The extravasation of blood is often confined to the grey matter, and consequently the sensations for pain and temperature may be lost to a greater degree than the sensation for touch.

The cells of the anterior horns are also liable to be damaged, with resulting muscular atrophy.

If the patient recovers, there is a likelihood of some spastic paralysis below the level of the lesion, and of some permanent wasting of the muscles which are supplied from the anterior horns at the level of the lesion; thus, for instance, wasting of the muscles of the arms is very liable to follow hæmorrhage in the region of the cervical enlargement. On the other hand, if the hæmorrhage is confined to the grey matter, there may be no paralysis below the level of the lesion.

Pathology.—There may be a single extravasation, or numerous hæmorrhagic foci may be present, and the condition of the surrounding cord largely depends on the existence or absence of previous disease. After injuries there is likely to be some hæmorrhage beneath the membranes as well.

Diagnosis.—The sudden onset of the symptoms suggests hæmorrhage. If the spine is injured the signs may be masked by the presence of extensive meningeal hæmorrhage. An important guide, when it can be demonstrated, is the loss of painful and thermic sensations, while the tactile sense is retained, thus pointing to a lesion of the grey matter. The existence of previous disease in the cord must be decided by the history.

Prognosis.—The prognosis depends on the cause and the extent of the hæmorrhage. In cases of injury where the hæmorrhage has been slight, patients often do well, although they may have permanent paralysis if cells with axons innervating important muscles be damaged.

Treatment.—The patient should be kept as quiet as possible; an ice-bag should be applied to the spine. The bowels should be opened. If necessary, morphia may be given to relieve pain. Ergot has been recommended for its supposed power to check the hæmorrhage.

CHAPTER XXV

TUMOURS OF THE SPINAL CORD

TUMOURS of the spine may be divided into two classes, according as they arise within or without the substance of the cord.

The chief tumours that arise *within* the cord substance (intramedullary tumours) are gummata, sarcomata, gliomata, and tuberculous growths. Probably the commonest of these intramedullary tumours are gummata.

Gliomata do not occur so often in the cord as in the brain, and intramedullary tuberculomata are decidedly uncommon.

Outside the cord (extramedullary tumours), sarcomata, carcinomata, psammomata, lipomata, fibromata, gummata, and hydatids are found: occasionally osteomata have been observed to press upon the cord. Carcinomata occur chiefly in the vertebræ, and are in most cases secondary to disease elsewhere.

Sarcomata generally arise from the pia mater, and are sometimes widely diffused over the membranes of the brain as well as of the cord. Their place of origin is then often difficult to determine, the disease at times appearing to have spread upwards from cord to brain, and at other times from brain to cord (Plate X.). In the latter case the extension may be due to infection of the cerebro-spinal fluid by a primary growth situated high up in the nervous system and exposed to the stream of cerebro-spinal fluid. (Stanley Barnes.)

Psammomata (dural endotheliomata) also grow from the pia mater (Fig. 63).

Lipomata usually grow from the areolar tissue outside the dura mater, but they may also arise on its inner surface.

Fibromata sometimes arise in connection with the nerve roots; reference to them will be found in Chapter X.

Gummata of the membranes are often ill-defined tumours, and are frequently accompanied by extensive meningeal infiltration and endarteritis.

Hydatids are important causes of compression of the cord. They nearly all grow in the areolar tissue between the dura mater and the bone. Of 44 cases tabulated by Schlesinger (quoted by Bland-Sutton), 39 were extradural and only 5 intradural.

Symptoms.—The symptoms vary somewhat according as the tumour is situated outside or inside the substance of the cord.

When outside the cord, a tumour produces symptoms by pressure (1) on the nerve roots and (2) on the cord itself.

The nerve roots generally suffer first, so that the patient complains of pain, which tends to

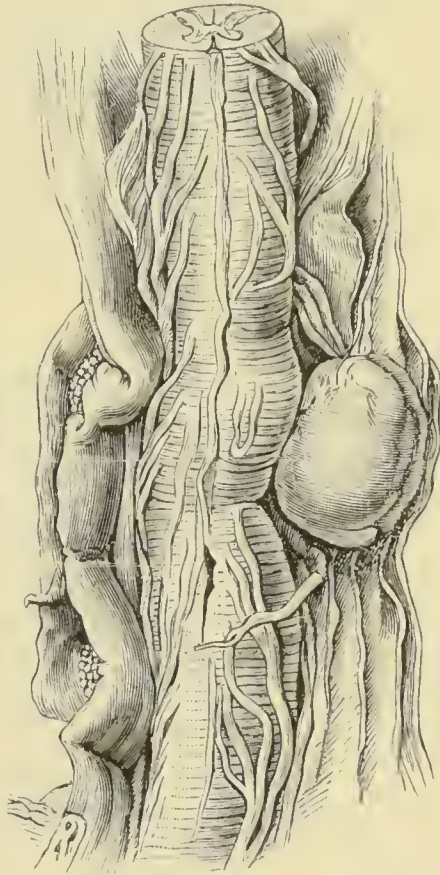


Fig. 63.—Portion of the spinal cord with a psammoma situated at the level of the intervertebral disc between the 10th and 11th thoracic vertebrae. From a woman 46 years of age. (Bland-Sutton; Museum, Middlesex Hospital.)

follow the root-distribution, and which for a time is generally unilateral. As the tumour grows, it presses on the cord and also perhaps on the anterior nerve roots, and thus to the pain are added weakness and loss of sensation.

The weakness below the level of the tumour will be of upper neuron type (spasticity, increased knee-jerks, extensor response of the plantar reflex, with no wasting or changes in the electrical reactions of muscles), while, at the level of the lesion, compression of one or more anterior roots may cause some paralysis and wasting over the area of their distribution.

The main alterations in sensation are anæsthesia over the areas supplied by the compressed posterior roots, to which may be added subsequently a more generalised anæsthesia below the lesion, due to pressure of the growth on the sensory columns of the cord. As a rule, the loss is more or less equal for all kinds of sensation. At the level of the tumour there may be a zone of hyperæsthesia. With increasing compression, a complete paraplegia develops with loss of power over the sphincters.

When the tumour grows in the substance of the cord (intramedullary) there is usually less pain, while paralysis and anæsthesia are more likely to be early symptoms.

The distribution of the weakness and of the alteration in sensation during the early stages of the disease depends upon the part of the cord in which the new growth originated. If, for instance, the substance of the grey commissure is destroyed, the signs will at first very probably simulate those of syringo-myelia, viz., loss of thermal and painful sensibility with preservation of tactile sensation. To these signs may be added some muscular wasting, as the pressure is felt by the cells in the anterior horns.

On the other hand, if the tumour grows for a

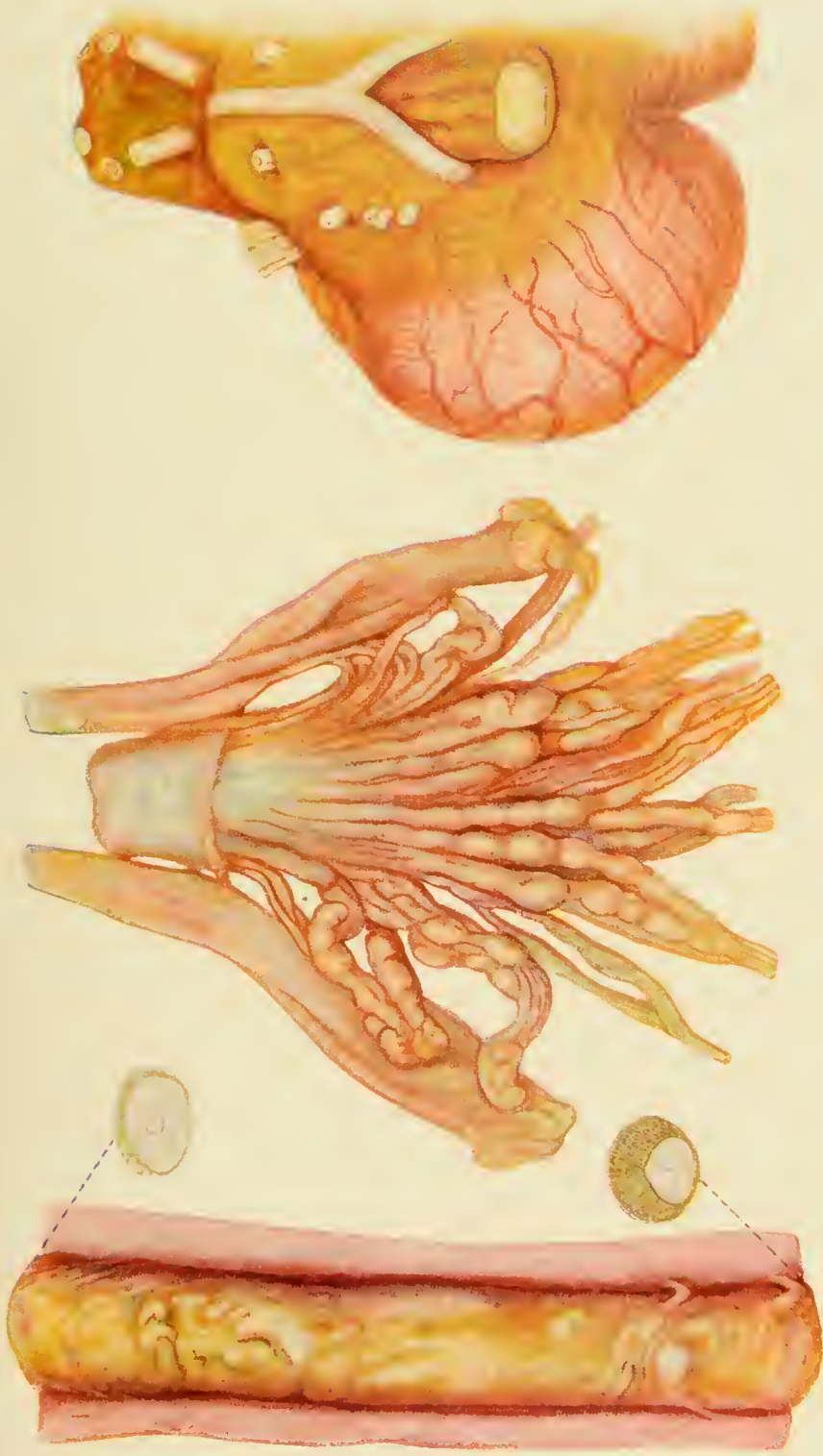


PLATE X.—Diffuse Sarcoma of the Brain and Cord.

(From an Illustration of a Case reported in the "*Transactions of the Pathological Society*" by
Drs. Coupland and Pasteur.)

time in one half of the cord, the signs will tend towards those of the Brown-Séquard form of paralysis, viz., loss of power below and on the same side as the tumour, with loss of sensation chiefly on the opposite side.

As the growth invades more and more of the cord, these early distinctions become masked by the onset of paraplegia associated with a variable amount of anæsthesia below the lesion.

The symptoms are then usually those of a transverse lesion of the cord, viz., spastic paralysis, anæsthesia, and loss of power over the sphincters below the lesion.

Tumours of the cauda equina.—The symptoms of tumours of the cauda equina, since the paresis is of the lower neuron type, are mainly flaccid paralysis with some wasting and electrical changes. The effect on the different structures varies with the level of the lesion. Thus the knee-jerks may be retained if the fibres going to the anterior crural nerve are unaffected, and in like manner the degree of sphincter control varies in different cases.

Diagnosis.—The recognition of tumours at an early stage is very difficult, for the pain is often indefinite, and, in the absence of other signs, is liable to be attributed to such conditions as muscular rheumatism, neuritis, and sciatica. Suspicion should, however, be aroused by the constancy of the pain, its tendency to follow an area of root distribution, and especially by the "girdle" character which it often assumes. These suspicions will be confirmed by the discovery of progressive weakness and sensory disturbance.

When the diagnosis of a spinal tumour has been made it is necessary to try to decide whether it is situated within or without the cord, for on that decision depends the possibility of its removal by operation.

In some cases the early implication of the

roots, subsequently followed by signs of gradual compression of the cord, are almost certain indications that the growth is extramedullary; in others, the dissociated anæsthesia, together with the absence of signs of pressure on the roots, makes it almost equally certain that the tumour is growing in the substance of the cord. But between these two groups there are many cases in which diagnosis is difficult, especially if the patient has already passed the early stages of the disease.

The next point to decide is the level at which the tumour is situated. The guide to this is to be found in the upper limits of the signs. When the anæsthesia and pain are of the root type their areas must be very carefully mapped out and identified with their particular roots, and the uppermost nerve root that is affected should demarcate the upper level of the tumour. Any local loss of power due to pressure on anterior roots can be utilised in a similar manner for localising purposes.

When the anæsthesia is of intramedullary origin, its upper level will likewise denote the position of the tumour, but any zone of hyperæsthesia that may exist about the level of the lesion must be taken into consideration, since its presence will indicate root-irritation, and will show the necessity of placing the growth rather higher than the anæsthesia indicates.

When the sensory signs are being taken as indicative of the level of the lesion, it must be remembered that the origin of the roots from the cord is not level with their foramina of exit, and that the spines of the vertebræ, owing to their obliquity, are not on a level with their corresponding bodies, otherwise there will be a likelihood of the localisation being made too low. The relations of the nerve roots to the vertebral spines are shown in Fig. 64.

Treatment. — The treatment of spinal

tumours should be similar to that recommended for those of the brain.

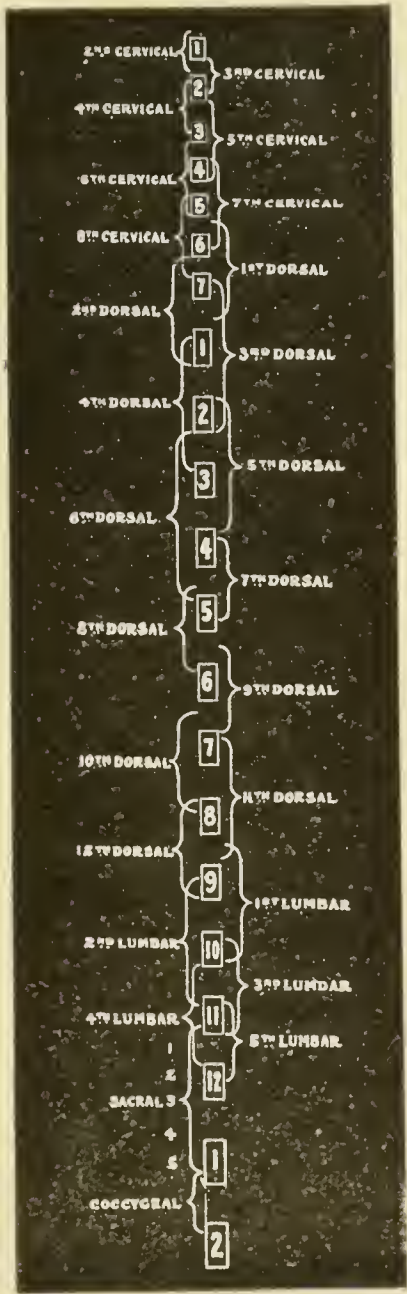


Fig. 64.—Diagram showing relations of the root origins of the spina nerves to the spines of the vertebrae. (After R. W. Reid, "Quain's Anatomy.")

If the tumour is syphilitic, or if there is a reasonable doubt regarding its nature, an efficient course of mercury and iodide should be tried. If the symptoms do not abate, the question of the removal of the neoplasm should be considered without delay. The possibility of its removal will depend upon its extramedullary situation, since tumours within the cord are inaccessible.

CHAPTER XXVI

SPINAL MENINGITIS AND MENINGEAL HÆMORRHAGE

SPINAL MENINGITIS

For purposes of classification, the cases of spinal meningitis may be divided into inflammations of the dura mater (pachymeningitis) and of the pia mater (lepto-meningitis), although it necessarily often happens that no distinction can be drawn between them either at the bedside or in the *post-mortem* room.

Pachymeningitis.—Inflammation of the dura mater may commence on the inner or outer surface, and the commonest causes are syphilis, injury, and extension of inflammatory processes from neighbouring parts, *e.g.*, from tuberculous disease of the vertebræ.

One form (pachymeningitis hypertrophica), in which the dura mater is greatly thickened, is especially apt to occur in the cervical region of the cord, and to cause wasting and pains in the arms. A rarer form is pachymeningitis hæmorrhagica, which is chiefly met with in asylums among general paralytics, and in which the exudation is chiefly blood.

Lepto-meningitis.—In lepto-meningitis the inflammation is at first seated in the pia mater and arachnoid.

Acute infections, as from tubercle, pneumonia, septicæmia, and epidemic cerebro-spinal meningitis, are the chief causes of this variety.

Symptoms.—The symptoms of spinal meningitis arise mainly from compression and irritation of the anterior and posterior nerve roots and also from extension of the inflammation to the structures of the cord. Hence the symptoms will differ with the position and intensity of the process. In hypertrophic meningitis of the cervical region, to which allusion has already been made, there is generally a slow strangulation of the nerve roots of the arms with consequent pains and wasting; and perhaps at a later date there may be signs of pressure on the cord giving rise to the effects of an incomplete transverse lesion.

In acute lepto-meningitis the signs of a general infection, such as fever, rapid pulse, and perhaps rigors are also usually present. The symptoms of irritation of the nerve roots are very prominent, and are shown by pain over the spine which radiates down the limbs, together with spasms, cramps, and rigidity of the muscles. The head is often drawn back, the back arched, and the abdomen retracted.

The control over the sphincters is frequently impaired, and there may be other signs that the inflammation is extending into the substance of the cord. Bed-sores and crops of herpes are very likely to occur.

In the more chronic forms, pain is generally a prominent feature and the motor symptoms are less severe.

In many of the infective cases, cerebral meningitis is also present, and to a considerable degree masks the spinal symptoms.

The principal characters of syphilitic meningitis are described in Chapter XXVII.

Diagnosis.—In its acute forms, spinal meningitis has to be distinguished from myelitis.

Reliance must be placed chiefly on the signs of irritation of the nerve roots which are so typical of meningitis, while paralysis is likely

to be more marked in a lesion of the cord. Of course the two conditions may be present at the same time.

In severe cases the symptoms may be suggestive of tetanus, but they are not in keeping with the onset of tetanus, which usually begins in the muscles of the jaw and neck.

The symptoms of a hemorrhage into the meninges are more rapid in their onset.

In the chronic cases the wasting of muscles may resemble that of progressive muscular atrophy, but the presence of the pains and anæsthesia generally show that the disease is not confined to the motor system. When the pains are the only symptoms their reference to the distribution of the nerve roots may deceive and lead to a diagnosis of muscular or abdominal trouble.

Examination of the fluid obtained by lumbar puncture may be an important aid to diagnosis, especially in the acute cases.

Prognosis.—The prognosis of acute spinal meningitis is not good, for the dangers of extension to the region of the medulla and brain and to the substance of the cord are all great. In a certain number of cases the symptoms subside, leaving behind them a variable amount of permanent damage, and in a few instances a good recovery takes place. In the chronic cases the outlook varies with the position and cause of the inflammation. Where the origin of the disease is outside the dura mater, as it is in spinal caries, the results are often very satisfactory if the original focus of disease is removed. Some of the syphilitic cases, of course, do very well, but, as mentioned elsewhere (p. 249), permanent cure even in these cannot be relied on.

Persistence or return of symptoms during treatment and extension of the disease are bad signs.

Treatment.—Whenever syphilis is suspected,

mercury should be administered. Potassium iodide may be given in full doses at the same time. Iodide is also undoubtedly sometimes useful in cases which apparently are not of syphilitic origin.

When the disease is chronic and has begun outside the dura mater, the pressure on the cord may sometimes be relieved by performing laminectomy.

In the acute infective cases, an ice-bag may be applied to the spine, though in many cases warm applications are more comforting. Counter-irritation is useful, but care must be taken not to set up bed-sores, especially if myelitis is present at the same time.

Mercurial inunction may be used, as in myelitis, for even though there is no history of syphilis, mercury seems often to exercise a beneficial influence over the inflammatory processes. Morphia must be given if required.

The same care in nursing and attention to the bladder are necessary as in the case of myelitis.

MENINGEAL HÆMORRHAGE

Etiology.—Hæmorrhage into the membranes of the cord may be associated with hæmorrhage into the brain, and is then usually of secondary consequence. Injury of the spine is sometimes accompanied by some hæmorrhage, which is more often outside the dura mater. Acute fevers, degenerated vessels, severe anæmias are occasionally the causes of hæmorrhages into the membranes of the cord as elsewhere.

Symptoms.—The symptoms are those of irritation of nerve roots and pressure on the cord: hence they are much the same as those of an acute spinal meningitis except that they arise more suddenly.

There is severe pain in the back and along the course of the nerve roots, accompanied by a

variable degree of spasm of muscles, and, when the cord is compressed, there is a more or less complete paralysis below the lesion. The deep reflexes are at first diminished, but later become exaggerated, and efficient control over the sphincters is often lost.

Prognosis.—The prognosis depends in the first place on the amount of blood extravasated and on the effects which it produces on the cord. The extent of the inflammatory and other after-effects remains to be seen, so that in every case the prognosis must necessarily be one of caution.

Diagnosis.—Hæmorrhage into the membranes of the cord is distinguished from meningitis by the suddenness with which the symptoms originate and by the absence of the more general symptoms that usually accompany acute inflammations of the cord. Otherwise they necessarily closely resemble each other.

The presence of an injury or some other distinct cause makes the diagnosis easier. It may be difficult sometimes to distinguish meningeal hæmorrhage from hæmorrhage into the substance of the cord, especially as both may be present at the same time. In the latter there is a tendency for the blood to be extravasated into the grey substance, and so to destroy sensibility to pain and temperature, leaving that to touch unimpaired.

Treatment.—If there are reasons to believe that the blood is compressing the cord, the tension should be relieved by performing laminectomy. In other cases, absolute rest should be enjoined, and an ice-bag should be applied to the spine. Calcium chloride may be given to promote clotting.

CHAPTER XXVII

SYPHILIS OF THE NERVOUS SYSTEM

THE effects of syphilis on the nervous system may be divided into the following groups:

1. Those due to *inherited syphilis*.
2. Those depending on the *direct results* of *acquired syphilis*, as evidenced by the presence of secondary or tertiary lesions.
3. Those depending on the *indirect results* of *acquired syphilis*.

INHERITED SYPHILIS

In some cases of idiocy and cerebral palsies of children, more especially the diplegias, the arrested development of the brain appears to be due to the inheritance of syphilis. It is also more than probable that congenital syphilis plays a part in the production of juvenile forms of tabes and general paralysis of the insane. Other rare forms of spinal degenerations have occasionally been attributed to congenital syphilis.

ACQUIRED SYPHILIS

Acquired syphilis is one of the most common causes of affections of the nervous system. More men suffer than women, and the symptoms are more often found in connection with the brain than with the cord, and even when the disease is most prominent in the cord there is generally some sign to suggest that the brain has not entirely escaped.

It is important to recognise that trouble may come on in a very short time after the occurrence of the primary infection, and less in connection with the nervous system even than in other organs can "secondary" and "tertiary" lesions be recognised, so far as any time limits are concerned.

In a very large number of cases the symptoms show themselves within the first two years after infection, and in many the first signs appear before the first twelve months have passed.

It is during the period to which "secondary" symptoms are usually allotted that the symptoms mostly begin.

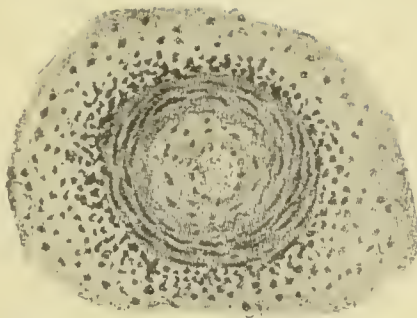


Fig. 65.—Endarteritis and periarteritis of a meningeal artery, from a case of syphilitic meningo-myelitis. (R. T. Williamson.)

The connective tissue and the vessels, veins as well as arteries, are the structures chiefly attacked, and the nervous tissue proper suffers in consequence.

Syphilis of the cord.—The most common lesion of the cord is one in which the membranes and nervous tissue suffer at the same time, and is known as meningo-myelitis, though, of course, either the membranes or the cord may be affected separately.

Meningo - myelitis.—The meninges become diffusely infiltrated with a gummatous substance over an area of varying extent, and at the same time the coats of many of the arteries which pass into the cord become thickened by an inflammatory process of a similar nature, which narrows

or altogether blocks their lumen. The meningeal thickening causes symptoms by compressing the nerve roots and other structures around, while the narrowed blood-vessels impair the circulation in the cord. The posterior arteries of the cord are generally most affected, and so the posterior columns and the pyramidal tracts especially feel the effects of the circulatory disturbances, with the result that the main symptoms are those of paraplegia, together with some degree of loss of sensa-



Fig. 66.—Section showing distribution of the anterior and posterior arterial system of the cord. The part shaded by dots shows the area supplied by posterior arteries of the cord. (R. T. Williamson.)

tion, to which some loss of control over the bladder is often added.

The symptoms, which are somewhat similar to those of other forms of myelitis, change for better or worse with alterations in the condition of the vessels, whereby more or less blood reaches the cord. In some cases, which Erb regards as forming a distinct group, the symptoms come on quite gradually, and are finally those of a partial myelitis, in which the motor columns suffer to a degree out of proportion to the sensory tracts, the control of the sphincters being also lost.

Acute syphilitic myelitis.—Sometimes syphilitic

changes in the cord come on very rapidly, and give rise to the clinical picture of acute transverse myelitis; indeed many cases included under this title are no doubt instances of rapid softening of the cord consequent upon syphilitic endarteritis and thrombosis.

Gummata.—Isolated gummata are not common either in the membranes or in the cord itself. When situated in the cord they are apt to produce the Brown-Séquard form of paralysis, or symptoms which closely simulate it.



Fig. 67.—Diagram showing division of a transverse section of the cord into three zones according to their arterial supply: (i.) Central zone (dotted) in the centre of the grey matter which is supplied by the anterior median or central arteries of the cord; (ii.) a peripheral zone (shaded) supplied by the peripheral arteries of the cord; (iii.) a third zone (pale) supplied by both central and peripheral arteries. (R. T. Williamson.)

Prognosis.—When the circulation to any part becomes almost arrested there is great danger of softening, followed by a sclerosis, which permanently damages the cord.

The patient is also very liable to relapse.

The course which the disease is going to take cannot be predicted until the results of treatment are seen. In some instances the symptoms disappear with great rapidity, in others there is permanent disablement from the effects of secondary

degenerations, in others the paraplegia becomes complete and complicated by bed-sores and cystitis, with subsequent death from exhaustion.

Diagnosis.—The combination of manifestations due to disease of the membranes and of the cord, together with the variability of the symptoms and the history of a primary infection, are some of the main points to which attention should be paid. A careful examination of the back should be made, in order to exclude the presence of caries, and the possibility of the existence of a tumour other than a gumma must be remembered.

Compression of the posterior roots by the thickened meninges may cause symptoms resembling those of tabes, but the rapidity with which the symptoms arise, together with the additional signs of meningitis and extension of the disease to the cord, generally make the diagnosis clear.

Syphilis of the brain.—In the brain, as in the cord, it is the vessels and the membranes that are specially liable to be attacked. The endarteritis is often followed by thrombosis and subsequent softening of brain tissue. A gummatous meningitis, through which the brain is damaged by pressure or extension of the inflammation, is the commonest affection of the membranes. Less commonly, definite discrete gummata are found.

Syphilitic meningitis very frequently begins at the base of the brain; the cranial nerves are soon damaged, and a special feature of the disease is the random way in which these are picked out. Optic neuritis is frequently present, as are other disturbances of vision due to pressure on the chiasma.

A gumma may, of course, give rise to the general and local signs common to other forms of cerebral tumour.

Variation of the symptoms, intense headache, worse at night, and early implication of the cranial

nerves are among the signs that suggest syphilitic meningitis.

Where the function of the frontal lobes is modified, the symptoms may closely simulate those of general paralysis of the insane, and the resemblance may be further intensified by the addition of convulsions, as the infiltration of the membranes extends over the motor areas. Irregular patches of infiltration, accompanied by symptoms which frequently disappear, may suggest the presence of disseminated sclerosis, the more so if at the same time patches of a similar kind are disturbing the functions of the spinal cord. In a doubtful case, time and the results of treatment must decide.

DISEASES DEPENDING ON THE INDIRECT EFFECTS OF SYPHILIS

These diseases are sometimes spoken of as parasyphilitic. The nerve degeneration by which they are characterised appears to be due to an impairment of vitality engendered by the syphilitic virus.

The two principal members of this group are tabes dorsalis and general paralysis of the insane.

~~General Infective Disease~~ ~~Local Disease~~ ~~Pia Mater~~ ~~Arachnoid~~ ~~Pachymeningitis~~
~~Invasion of Local Disease: dura mater - Pachymeningitis~~
 General Infective Disease: - pia mater arachnoid, Lepto meningitis
 Invasion of Local Disease: dura mater - Pachymeningitis

SECTION V.—ORGANIC DISEASES OF THE BRAIN

CHAPTER XXVIII

MENINGITIS

INFLAMMATION of the membranes of the brain may occur as part of a general infective disorder or from extension of some local disease of the skull. In the latter case the dura mater is especially liable to be affected (*pachymeningitis*), but in the former the primary seat of the inflammation is in the pia mater and arachnoid (*lepto-meningitis*).

The micro-organisms which most frequently invade the meninges are those of tubercle, pneumonia, typhoid, septicæmia, cerebro-spinal fever, and its sporadic form of posterior basic meningitis. In many cases the primary infective organism becomes associated with streptococci and staphylococci during the course of the illness.

While the invasion of the meninges may be the predominating feature during life, an examination of the body after death very frequently reveals trouble in other organs as well. Thus tuberculous meningitis is practically always a part of a more generalised infection, as also are the cases due to the organisms of typhoid and, though perhaps less often, of pneumonia. The syphilitic forms have been already mentioned.

I.—TUBERCULAR MENINGITIS

Etiology.—Tubercular meningitis is most commonly found in children from two to ten years of age, though its occurrence in adults is by no means rare.

The contributory causes are debility and the presence of tuberculosis of the lungs, glands, or some other part of the body.

Symptoms.—The early symptoms vary according to (1) the intensity of the general toxæmia and (2) the prominence or otherwise of local symptoms.

As a rule, there are signs of general disturbance for some considerable time before there is any reliable evidence of cerebral trouble, the child being irritable, languid, and generally out of sorts. An early sign of considerable importance is vomiting for which no adequate cause can be found. The temperature is a little raised at night, and often runs rather an irregular course throughout the illness.

vomiting
for which no
cause can be found

In other cases, distinct cerebral signs occur at an earlier date, and severe headache, with perhaps a squint and a general convulsion, may be among the symptoms to which attention is first attracted, though here again careful inquiry often elicits the fact that the child has not for some time previously been in its usual health.

headache
Squint
Convulsions

The symptoms can best be remembered by considering what effects the pathological changes produce upon the various parts of the brain. The small, grey, semi-translucent tubercles are situated in the pia mater, and are distributed chiefly over the base of the brain. They are very prone to occur along the course of the middle cerebral artery, and are generally most easily recognised between the opercula of the Sylvian fissure. They are usually much less numerous over the convexity of the brain, but in this respect their distribution is very variable in different cases.

Their growth is accompanied by a serous exudate of varying density. The chief effects produced by these processes are:

(1) Irritation and compression of different structures of the brain.

(2) General rise of intracranial pressure due to the exudation of fluid and the limitation of its circulation by the adhesion of the inflamed parts, and especially by the blocking of the foramen of Majendie and the consequent distension of the cerebral ventricles.

The *local* effects of the irritation and compression are chiefly felt by the cranial nerves, and may be roughly enumerated as follows:

Olfactory nerve.—Usually no definite sign of impairment.

Optic nerve.—Optic neuritis is common and a useful aid to diagnosis. During an examination of the eyes for evidence of optic neuritis, the possible presence of tubercles in the choroid should be remembered.

Third, fourth, and sixth nerves.—These three motor nerves of the eye may be considered together. One or more are often affected, with resulting squint and ptosis. The pupils may be unequal and reaction to light lost or impaired.

Trigeminal nerve.—There are not usually any definite signs connected with the fifth nerve, but, if present, they will take the form of pain in the area of its surface distribution and of spasm or paresis of the muscles of mastication.

Facial nerve.—Extension of the inflammation to the trunk of the facial nerve gives rise to weakness of a part or whole of one side of the face. Twitching of the facial muscles may also occur, if irritation be present.

Auditory nerve.—Deafness results from implication of the auditory nerve. It is not usually a marked symptom, for the impaired mental condition of the patient generally overshadows it.

Glosso-pharyngeal nerve.—There is no obvious symptom connected with the glosso-pharyngeal.

Vagus.—During certain stages of the disease irritation of the vagus may show itself by the slow beating of the heart, but paralytic signs are

usually the more prominent, and the heart is rapid and irregular. The respiratory rhythm is also altered, but the functions of both heart and respiration are in all probability largely modified by the more general effects of the disease upon other parts of the brain, and the variations of the respiratory rhythm will be again considered under that heading.

Spinal accessory nerve.—Irritation of the spinal portion of the spinal accessory is perhaps responsible to some extent for the retraction of the head, though probably this is also influenced by irritation of the cerebellum, but retraction of the head is often temporary and, in many cases, not a prominent feature of this form of meningitis, which is in keeping with the fact that the exudation is frequently limited chiefly to the area of the circle of Willis, and does not extend sufficiently far down to implicate the cerebellum and the cervical nerve-roots.

Hypoglossal nerve.—There are not usually any signs of disturbance of the function of the hypoglossal nerve.

The convexity of the brain.—When the inflammation extends on to the convexity of the brain there may be monoplegia or hemiplegia. Localised convulsions may also be a prominent symptom.

When we come to consider the effect of the *general increase of intracranial pressure*, we find a number of symptoms referable to compression and irritation of the cortex and meninges.

Pain.—The headache, perhaps slight in the early stages, may become so intense as to cause the intermittent shrieking known as the "hydrocephalic cry."

Vomiting.—Early in the disease the vomiting may be due to the general toxæmia, but in the later stages it is mainly the result of the rise of intracranial pressure, as in the case of cerebral tumours.

The respiratory rhythm is altered. Sometimes it is of the Cheyne-Stokes type, but more often a few respirations of equal intensity are succeeded by a short pause. The breathing is nearly always rapid.

The pulse for the most part is quickened, but in the earlier stages of the illness it may be slow from irritation of the vagus, as already mentioned.

Convulsions are common. They arise from extension of the tubercles on to the cerebral cortex or from irritation induced by the general rise of pressure; localised twitchings also commonly occur.

The abdomen is retracted, and known as the "scaphoid abdomen."

There are pronounced mental symptoms: the patient is irritable, lies curled up on his side, avoids the light, and shows disinclination to answer questions or to be troubled in any way.

The vasomotor system is deranged, and if the finger-nail be drawn over the skin, it is rapidly followed by a flush that is known as the tâche cérébrale.

The temperature throughout the illness is somewhat irregular, and not generally very high; but towards the end there is often a considerable rise, which may continue for some time after death.

Kernig's sign.—The effects of cerebral irritation are seen in the increased "tone" (hypertonus) of muscles, and it is on this fact that Kernig's sign depends (Fig. 68).

To obtain the sign the patient should be lying on the back, and, care being taken to keep one leg flat on the bed, the other should be flexed at the hip and knee. When the tone of the muscles is increased, the degree of extension which it is possible to obtain at the knee-joint while the thigh is flexed upon the abdomen is considerably less than normal. Kernig's sign is said to be present in over 80 per cent. of the cases of meningitis. It is, therefore, of considerable diagnostic

significance, but it may, of course, be associated with other forms of cerebral irritation.

General course of the disease.—Attempts have been made to divide the disease into stages of irritation and compression, but the two conditions are necessarily concurrent, and it is very seldom that any sharp line can be drawn between them.

The duration of the symptoms is very variable. Death may occur within a week or ten days from

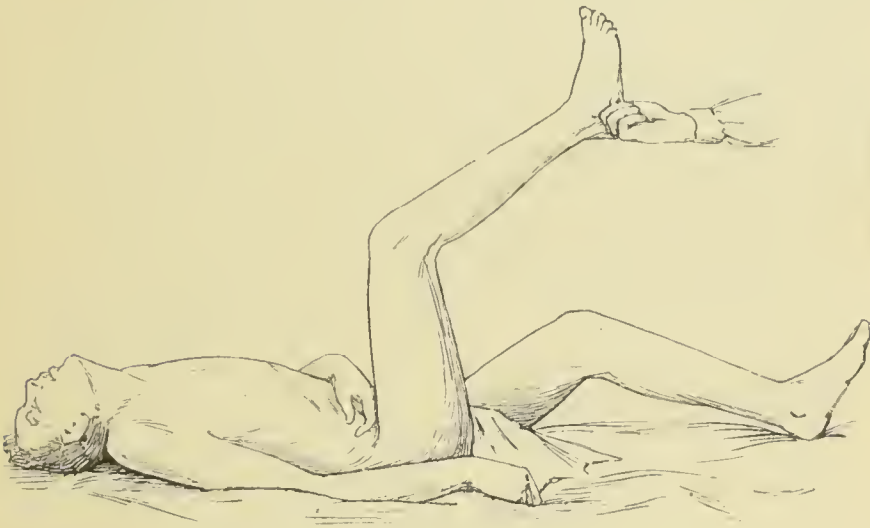


Fig. 68.—Method of obtaining Kernig's sign.
(After a photograph by Dr. W. Thyne.)

the onset of acute symptoms, or it may be postponed for three weeks or more.

Prognosis.—There is no evidence to lead us to hope that a case of undoubted tuberculous meningitis is likely to recover. The prognosis is bad, and all cases in which the symptoms are those of generalised meningitis are practically hopeless. Moreover, as already insisted upon, the meningeal inflammation is often only the more prominent part of a widely spread infection.

Diagnosis.—In children the chief difficulty in arriving at a diagnosis exists during the early stages of the disease. The child has perhaps been

ailing for some time, but gets worse so gradually that it is difficult to assign the correct value to the vague symptoms of headache and gastric disturbance which so often precede those of a more definite character.

In other cases the difficulty is of the opposite kind, for the symptoms come on so acutely that for a short time it appears more likely that an acute specific fever is about to develop. Between these two extremes there is every gradation, so that it is nearly always difficult to make a diagnosis with any certainty at an early stage of the disease.

Inflammatory troubles of the middle ear, the mastoid, and other parts of the skull may for a time be mistaken for meningitis, as also may the mental disturbance sometimes due to worms.

In adults the distinction between typhoid fever and tuberculous meningitis is often difficult. The main points to be relied upon can be tabulated as follows:

	TYPHOID.	MENINGITIS.
HEADACHE ...	Early symptom; tends to disappear later	Headache more intense; gets worse during the course of the illness. <u>Hydrocephalic cry.</u>
OPTIC NEURITIS	Not present ...	Frequent.
EHRlich's REACTION	Generally obtained...	Exceptional, but may be present.
WIDAL'S REACTION	Usually obtained at the end of ten days	Not present.
RASH ...	Typhoid spots appear in crops from seventh to tenth day and onwards.	Not present.
SQUINT ...	Not present ...	Very frequent.
TUBERCLE IN OTHER PARTS	Absent ...	Suggestive, though of course not necessarily diagnostic.
TEMPERATURE	Fairly regular course	More irregular.

Difficult cases can only be diagnosed by a careful review of all the symptoms in relation to one another, for nothing is more frequent than for exceptional and deceptive signs to appear in both these diseases.

The delirium of lobar pneumonia, especially of the apical form, is sometimes very deceptive, and the knowledge that the pneumococcus may invade the meninges as well as the lungs makes the diagnosis still more uncertain in doubtful cases.

When the case appears to be one of meningitis a careful search must be made for other causes before finally concluding that it is tuberculous. The ears must be examined for signs of suppuration, and any possibility of a cerebral abscess must be carefully considered. It is most important not to overlook any of these other causes of meningitis, since they may often be relieved by surgical treatment when the operation is undertaken in time.

Lumbar puncture may be an aid to diagnosis. In tuberculous meningitis the fluid is generally clear or only slightly flaky. Well-marked lymphocytosis is usually present, and in some cases tubercle bacilli can be detected by staining after centrifugalisation.

Treatment.—From the foregoing paragraphs it may be gathered that no drugs have any specific influence on the disease. Mercury has occasionally been observed to produce an improvement, and it may be given by inunction. Potassium iodide is useless in tuberculous meningitis, but should, of course, be at once given if there is any suspicion as to the affection being syphilitic. Relief of pressure is sometimes obtained by tapping the spinal membranes, and in this way a temporary amelioration of the symptoms may be secured, but the adhesions at the base of the brain usually prevent the drainage being sufficiently free to give any permanent relief.

The following general measures should be adopted: The patient should be kept in a dark room, his head shaved, an ice-bag applied, and plenty of nourishment given. Phenacetin, anti-pyrin, and salicylates, and in severe cases opium, may be given for the relief of headache. The bowels should be kept open.

Signs of heart failure will indicate the necessity for strychnine, digitalis, and other suitable stimulants.

II.—EPIDEMIC CEREBRO-SPINAL MENINGITIS (SPOTTED FEVER)

Etiology.—Epidemic cerebro-spinal meningitis (cerebro-spinal fever or spotted fever) is most prevalent among children and young adults, but is very rarely found in infants under a year.

The micro-organism is the diplococcus intracellularis of Weichselbaum, commonly spoken of as the "meningo-coccus," and is identical with that found in cases of posterior basic meningitis, which latter must be regarded as sporadic cases of the epidemic form.

The exact conditions which cause the disease to assume an epidemic character are unknown, but probably atmospheric changes, overcrowding, and dirt have all a share in modifying the virulence and prevalence of the organism.

The disease is undoubtedly contagious, and, although not markedly so, it appears in some instances to have been conveyed by a third person. The organism does not seem to possess great vitality outside the body, and frequently is almost entirely confined to parts which have no communication with the exterior. It is, however, often to be found in discharges from the ear and nose and in the expectoration of patients, and therefore the utmost care must always be taken to prevent the disease from spreading in this way.

The micro-organism is not very easy to culti-

vate, and grows best on serum. During life it can be obtained from the spinal fluid by lumbar puncture, but in many instances the results of bacteriological examination of the cerebro-spinal fluid are negative.

Pathology.—The meninges, brain, cord, and peripheral nerves (both cranial and spinal) are all apt to be affected. The pia and arachnoid membranes are the chief seat of the infection, and the exudation, which may be serous or purulent, is generally most intense at the base of the brain, although it often extends some distance over the cerebral hemispheres and the lobes of the cerebellum. In the most acute cases, nothing more than intense injection of the pia mater may be seen with the naked eye, but microscopic examination generally shows that a considerable amount of exudation has already taken place.

In the chronic cases the membranes are often very much thickened, and have contracted adhesions which may interrupt the circulation of the cerebro-spinal fluid and lead to hydrocephalus.

Similar changes are found in the pia mater and the arachnoid of the cord, more especially on its posterior surface.

Patches of softening and hæmorrhages are often present in both the cerebral and spinal substances.

The inflammation is also very apt to extend along the cranial and spinal nerves, which become swollen and softened in consequence.

It is stated that the second, fifth, seventh, and eighth are the most liable to suffer, and that inflammatory changes of an advanced character can generally be found in the Gasserian ganglia.

Symptoms.—The onset is usually very sudden, and of the early symptoms the most prominent are vomiting and pain, the latter being either localised to the head or of a generalised character.

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8.

The presence of meningitis of the brain and cord is soon shown by palsies of the cranial nerves, which cause strabismus, ptosis, unequal and fixed pupils, facial paralysis, and deafness; by rigidity of the neck and trunk muscles, with retraction of the head and opisthotonos; by optic neuritis, convulsions, hydrocephalic cry, Cheyne-Stokes respiration, delirium, and coma; and sometimes by hemiplegia and paraplegia. The mechanism by which these symptoms are produced *i.e.*, by irritation and compression, has already been discussed in the section on tuberculous meningitis, to which the reader is referred.

Inflammatory conditions of the eyes, nose, ears, and lungs are often present. The eye symptoms are very important, and include conjunctivitis, iritis, corneitis, and sometimes inflammation of all the structures of the eyeball, which may result in permanent blindness. Apart from these changes, a temporary loss of vision may occur. Disease of the ears is a complication frequently met with; the internal ear may be damaged by the extension of the inflammation along the auditory nerve, to which reference has already been made; and there is often acute inflammation of the middle ear, in the discharge from which the specific micro-organisms may be found.

Not unfrequently there is a discharge from the nose, from which the diplococci may also be isolated.

The organism may also reach the lungs, and produce broncho-pneumonia.

The skin.—Small hæmorrhages into the skin, forming petechial spots, are frequently present; hence the name "spotted fever." Herpes has also been frequently observed.

The *pulse* and *temperature* are very variable. Swelling of the *joints* sometimes occurs.

Course of the disease.—In the fulminating cases, death may take place within a few days.

The ordinary acute cases generally exhibit all the main symptoms that have been detailed, and when they are about to terminate fatally the coma deepens and all power over the sphincters is lost, as in other forms of meningitis.

Sometimes the disease runs a more *chronic* course, and is accompanied by great emaciation and weakness, and—if recovery takes place—by impairment of the mental faculties or hydrocephalus.

Cases in which there are intermission and remission of symptoms are sometimes observed.

Diagnosis.—Differentiation has to be made between this disease and other forms of meningitis, such as those due to the tubercle bacillus and the pneumococcus.

To decide the diagnosis, lumbar puncture should be performed, and the fluid thus obtained should be examined for the diplococci. The presence of these makes the diagnosis conclusive, but too much importance must not be attached to a negative result, for the organism is cultivated with difficulty, and may be absent during the remissions and also in the later stages.

Prognosis.—The death-rate varies in different epidemics according, presumably, to the virulence of the organism. In individual cases the prognosis must be based upon the intensity of the general infection and upon the signs denoting the degree of intracranial pressure.

Treatment.—There is at present no special form of treatment beyond that which is applicable to the other varieties of meningitis, but there is some reason to hope that an antidotal serum may be procurable in the near future.

An attempt to relieve the intracranial pressure may be made by performing lumbar puncture, and in some cases this procedure, repeated at short intervals, appears to assist materially in tiding the patient over the critical period of the disease.

III.—POSTERIOR BASIC MENINGITIS

Etiology.—In this form of meningitis the inflammation is mainly confined to the posterior part of the base of the brain. The micro-organism, first noted in connection with this disease by Still, has proved to be identical with the *Diplococcus intracellularis* of epidemic cerebro-spinal meningitis, of which posterior basic meningitis must be considered to be a sporadic form.

The organism probably reaches the membranes of the brain by way of the Eustachian tube and middle ear, and any debilitating process, such as rickets, injury, or catarrh, renders the child more liable to infection. Children of both sexes are attacked, and the majority of cases occur during the first year of life.

Pathology.—The exudate is found over the posterior part of the base of the brain, and, if it has existed long enough, there may be occlusion of foramina in connection with the fourth ventricle and interruption of the flow of cerebro-spinal fluid; the latter then collects in the lateral ventricles and produces hydrocephalus. Sometimes the inflammation extends some way down the cord or on to the vertex of the brain.

Symptoms.—The onset is usually acute, but the symptoms may be indefinite for a time. Vomiting occurs early, and is generally accompanied or quickly followed by retraction of the head, which is one of the most prominent features of the disease (Plate XI.).

More widely spread tonic spasms cause general rigidity, or even opisthotonos; convulsions may also occur. The rise of intracranial pressure is shown by the raised fontanelle, and it is probably the cause of the temporary blindness which is often present, since optic neuritis is seldom met with. The condition of the pupils varies; in some cases they are contracted, in others dilated. Owing to retraction of the upper lids, the eyes



PLATE XI.—Case of Posterior Basic Meningitis showing Extreme Retraction of the Head.

(From a Sketch by Dr. Penrose Williams.)

often appear unduly prominent; nystagnus may sometimes be observed. Strabismus often occurs, though not with such frequency as in tuberculous meningitis.

Alterations resembling those of tuberculous meningitis may take place in the pulse and respiration.

The deep reflexes vary in their reactions, and are sometimes increased, sometimes absent.

In the later stages the mental powers may be impaired by hydrocephalus.

Diagnosis.—At the onset it is often difficult or even impossible for a time to decide whether meningitis or some other acute febrile condition, *e.g.*, pneumonia, is present. After a few days the reference of symptoms to the head, together with the absence of signs of a generalised infection, makes the locality of the disease more apparent, and it is then necessary to exclude the presence of sinus thrombosis, otitis media, and mastoid disease before arriving at the diagnosis of meningitis. When the diagnosis of meningitis has been made it is still necessary to attempt to decide the nature of the infection, and the chief difficulty lies in differentiating the posterior basic from the tuberculous form.

Reference to the list of symptoms shows that in posterior basic meningitis the rigidity and retraction of the head are the most prominent symptoms, while optic neuritis, palsies of cranial nerves, and retraction of the abdomen are less likely to be present. The detection of tubercles in the choroid will, of course, decide the question, as also may a bacteriological examination of the cerebro-spinal fluid. A blood-count may also be of value, for in posterior basic meningitis leucocytosis is usually greater than that which occurs in tuberculous meningitis.

Prognosis.—The prognosis of posterior basic meningitis is not altogether unfavourable. A

early.
acute febrile condition
after.
Sinus Thrombosis
Otitis media
Mastoid Dis

considerable number of patients get well, though often with mental impairment, the result chiefly of the attendant hydrocephalus. The duration of the illness varies, but often lasts several weeks, and thus may be longer than in the tuberculous form.

Treatment.—Mercury may be given on account of its general tendency to influence inflammatory processes, and it is well borne by children. Iodides have also been recommended. Operations with a view to draining off the excess of fluid and relieving the pressure have not met with any constant success, but cases have been recorded in which repeated lumbar punctures seem to have been followed by beneficial results.

IV.—VERTICAL (SIMPLE) MENINGITIS

A vertical form of meningitis has been described by Dr. Lees and Sir Thomas Barlow.

The inflammation is most marked over the anterior part of the brain, and when it is limited to that region the diagnosis both of the meningitis and of the nature of the infection is naturally difficult.

Moreover, its signs are apt to be masked by coexisting lesions, such as pericarditis and pneumonia. The symptoms of irritation of the cranial nerves and retraction of the head are usually absent, but convulsions are likely to arise from extension of the inflammation to the motor areas of the cortex. The prognosis is not good, and the condition often appears as a complication towards the terminal stages of other illnesses.

The duration of the disease is usually shorter than that of both the tuberculous and posterior basic forms of meningitis.

V.—SUPPURATIVE MENINGITIS

Etiology.—The invasion of the meninges by pyogenic organisms may take place from extension of local suppuration, or it may be a part of the

terminal stage of a general infection. The local conditions which most frequently give rise to meningitis are suppurative otitis media, mastoid disease, and cerebral abscess, all of which are often closely associated with one another.

The **symptoms** of suppurative meningitis do not materially differ from those described under the heading of tuberculous meningitis, since they also depend upon the irritation and pressure of exudation. The general tendency, however, is for the suppurative forms to spread over the convexity of the brain rather than to remain confined to the base; hence the functions of the cranial nerves are apt to be less extensively deranged than in the tuberculous form.

Suppurative meningitis is accompanied by severe general symptoms, and when diffused always causes death.

The **treatment** consists in relieving the symptoms on the lines already mentioned. When the meningitis is once established the opportunity for surgical interference has passed, and it is, therefore, most important to deal promptly with any local suppurative process about the skull before any extension to the membranes can take place.

VI.—PACHYMENINGITIS EXTERNA

In some cases where there is direct extension from the bones of the skull the suppuration is for a time, at any rate, confined to the outer surface of the dura (pachymeningitis externa), and if a collection of pus be localised, incision and drainage may be attended with success. Left to itself, the natural tendency is for the pus to infect the inner membrane.

CHAPTER XXIX

CEREBRAL PALSIES OF CHILDREN; AMAUROTIC FAMILY IDIOCY; HYDROCEPHALUS

CEREBRAL PALSIES OF CHILDREN

THE paralysees of cerebral origin which occur in childhood may be grouped, according to their causation, as follows:

A—*Before birth: e.g.*

- (1) Primary arrest of development.
- (2) Changes depending on maternal syphilis or some other toxic condition.

B—*During birth: e.g.*

- (1) Hæmorrhages into the meninges.
- (2) Direct injury of cerebral cortex.

C—*After birth: e.g.*

- (1) Acute encephalitis.
- (2) Thrombosis.
- (3) Meningeal hæmorrhage.
- (4) Meningitis.

Pathology.—As might be expected from the variety of the causal lesions, the brains differ very markedly from one another in their appearance.

In some, an abortive development of the motor areas is the main feature—a condition known as cerebral agenesis. In others, the brain is small, and exhibits a generalised sclerosis; while in others again, cystic conditions, known as porencephaly, are found.

Thrombosis of vessels and softening of brain tissue occur from encephalitis, and may give rise

later on to a sear, over which the membranes may be thickened and adherent.

The paralysis usually takes the form of a hemiplegia or a diplegia.

CEREBRAL DIPLEGIA

Cerebral diplegia is characterised by spasticity of both sides of the body, and by a variable degree of weakness. Arrested development of the motor areas during intrauterine life appears to be the most common cause. The nerve elements which should make up the motor areas of the brain either fail to develop, or degenerate during the period of their development, poisoned by syphilis or some other toxin. In some cases they appear to atrophy from sheer want of vitality, and so form a connecting link with those in which the degeneration occurs in later childhood, *e.g.*, in Friedreich's disease and some of the myopathies, a connection which is further emphasised by the occasional tendency of cerebral diplegia to appear in different members of the same family.

All gradations exist between complete absence of the motor areas and slight impairment of their function, with corresponding clinical variations. When the damage to the brain occurs during the birth of the child it is most probably due to a hæmorrhage into the meninges whereby the motor areas of the legs are more or less symmetrically injured, the lesion sometimes extending as far over the hemispheres as the areas corresponding to the movements of the upper extremities.

After birth the injuries are far more likely to be unilateral, and to cause hemiplegia rather than diplegia.

It very frequently happens that the exact period at which the damage occurred cannot be ascertained with any degree of certainty from the history of the case.

Symptoms.—The main symptoms of cerebral

diplegia are rigidity, weakness, involuntary movements, and mental infirmity, but their variability in degree is very great, so that cases differ very considerably from one another.

In a typical case of cerebral diplegia it is noticed that the child's limbs are stiff, and that the movements are not as free as they should be.



Fig. 69. — "Scissors-legged" progression in a case of cerebral diplegia.

On examination, there is evidence that the child has the signs of degeneration of the motor tracts, as shown by weakness, rigidity, and increased knee-jerks. The behaviour of the plantar reflex is not of much assistance in young infants, since in them it is normally of the extensor type, but in older children it possesses the usual significance.

As a rule, the rigidity is rather out of proportion to the weakness, and, on the patient attempting to walk, the well-known "scissors-legged" attitude is assumed, owing to spasm of the adductors of the thigh (Fig. 69). The arms share in the rigidity, though oftentimes to a less degree. The face may also be affected, and occasionally there is atrophy of the optic nerve.

The weakness, always present in some degree, is often rather masked by the rigidity, which makes voluntary movements far more difficult than they would otherwise be.

Involuntary movements occur in a considerable number of cases, and often attack the hand and arm (less commonly the leg and face), taking the form known as athetosis, the chief characters of



PLATE XII.—Instantaneous Photograph showing Bilateral Athetosis
in a Case of Cerebral Diplegia.

(From a Case under the care of Dr. George Ogilvie.)

which are slow, more or less rhythmical, "squirming," involuntary movements of the fingers and thumbs, in which hyperextension is a prominent feature. Sometimes the whole arm takes part in the movements, which may be so violent and uncontrollable as to necessitate strapping the limb to the side. Other movements may take the form of tremors, or may be of a more irregular choreiform nature (Plate XII.).

All degrees of mental impairment are met with, from "backwardness" to complete idiocy. Articulation also is often impaired.

Epileptic fits are often associated with cerebral diplegia.

The less severe cases of diplegia, in which the symptoms are confined to the legs, and consist of some rigidity combined with weakness of only moderate degree, are often classed together in a group under the heading of Little's disease, after the name of the surgeon who first described them.

Diagnosis.—When the symptoms are pronounced, there is but little difficulty in recognising their nature.

The limbs of children suffering from severe rickets or scurvy rickets sometimes appear to be paralysed, but the absence of rigidity, with the enlargement of the ends of the long bones, and other signs of rickets, show that the weakness is not of nervous origin.

In older children, with a moderate degree of symptoms, disseminated sclerosis and Friedreich's disease may require consideration. Disseminated sclerosis is very rare in childhood, and possibly some of the cases recorded under that heading have been, in reality, cerebral diplegias.

Variability of symptoms and nystagmus may help to distinguish difficult cases, and, of course, the history, when obtainable, is a most reliable aid.

It is important to differentiate the two diseases

carefully, since there is ultimately no great hope for those suffering from disseminated sclerosis, whereas the slighter forms of cerebral diplegia often tend to improve.

Friedreich's disease comes on later in life, and the ataxy, loss of deep reflexes, nystagmus, and other characteristic symptoms will generally enable it to be distinguished.

Prognosis and treatment.—Extraordinary improvements can be effected in the less severe cases by patiently persevering with mental and physical education, and for the latter carefully regulated gymnastics should be prescribed. Pronounced mental impairment and frequent fits are of evil significance, and the onset of athetotic movements usually indicates that the chance of further improvement in the use of the limbs is but slight.

INFANTILE HEMIPLEGIA

The hemiplegias of childhood are, for the most part, due to injuries received at the time of birth or to post-natal inflammatory and vascular lesions.

During prolonged and difficult labours, hæmorrhage or some other injury may occur, by which the functions of the cortical motor areas are impaired.

After birth, certain blood states, especially those brought about by specific fevers, are conducive to thrombosis in the cerebral vessels, or the direct invasion of the brain tissue by micro-organisms may give rise to an acute encephalitis, analogous to the lesion in acute anterior poliomyelitis.

Symptoms.—Of the cases due to encephalitis, most occur in children between the ages of three and six, and the onset is generally marked by fever, malaise, and convulsions, with more or less disturbance of consciousness.

Between the convulsions that attend the onset of the disease, or more frequently after the lapse

of one to several days, unilateral weakness is observed, generally most pronounced in the upper extremity; and in some cases the motor nuclei of cranial nerves are affected like those of the cord in anterior poliomyelitis.

Sometimes the power gradually returns in the affected parts, but in the majority of cases a considerable amount of paralysis persists. The later symptoms are, in the main, those already described in the diplegias, viz., fits, mental deterioration, and, perhaps, athetosis.

When the lesion is in the left cortex there is the possibility of aphasia, but the right side will probably take up the function of speech if the subject is a child.

Diagnosis.—The convulsions and distribution of the paralysis indicate a cerebral, as opposed to a spinal, lesion, while fever, and drowsiness or complete loss of sensibility, are the usual accompaniments of cases of acute encephalitis. The presence of a valvular lesion of the heart will suggest the possibility of embolism.

When a long-standing case is seen for the first time, its cerebral nature will be evident from the spasticity, increased reflexes, and absence of electrical changes, in contrast to the flaccidity, loss of reflexes, and the reaction of degeneration found in the paralysis following acute anterior poliomyelitis of the cord. The atrophy is much more evident in cases following the latter lesion, although the limbs of the infantile hemiplegic also look wasted owing to arrest of their development, and club-foot is a common occurrence in both diseases.

Treatment.—During the febrile period of encephalitis the child should be kept quiet in bed and the room should be darkened. A mixture containing bromides and a purge of calomel or grey powder should be given, and the diet should be as light as possible. When the signs of cortical

irritation have quite disappeared, the most careful and persistent attention must be given to the paralysed limbs. Passive and, where possible, active movements must be practised regularly, and every possible effort should be made to prevent the onset of contractures.

The ultimate results in many instances are surprisingly good, but no accurate prognosis can be given for any individual case, since the onset of fits, athetosis, or permanent mental disability may mar the most promising outlook.

When fits occur they must be treated with bromide, as in ordinary epilepsy.

Tenotomy may be useful in both hemiplegia and diplegia if contracture is hampering a movement that would otherwise be useful.

CEREBELLAR PALSIES OF CHILDHOOD

The causes of the cerebellar lesions of childhood are very similar to those producing the cerebral palsies, and may, in like fashion, be divided into those occurring—

A—Before birth.

B—During birth.

C—After birth.

The main symptom in cases of cerebellar disease in children is ataxia, and F. E. Batten* has shown that cases of ataxia in childhood in relation to cerebellar disease may, from a clinical point of view, be classified as follows:

- (1) Congenital cerebellar ataxia: in which the ataxia has been noticed from earliest life.
- (2) Acute cerebellar ataxia: due to encephalitis of the cerebellum, in which the ataxia has developed during the course of or after some acute illness in a child who was formerly healthy.

* *Brain*, 1905, Pts. cxi., cxii.

- (3) *Progressive cerebellar ataxia*; in which ataxia has gradually developed in a child who has previously been healthy and of normal development.

The value of this classification is enhanced by F. E. Batten's observations on the *prognosis*, which lead him to conclude that the cases which come under the heading of congenital ataxia, and those also which are due to encephalitis, show a tendency to improve gradually, while in those belonging to the group of progressive ataxia the outlook is bad.

AMAUROTIC FAMILY IDIOCY

The results of an extreme degree of cerebral degeneration in childhood are seen in the condition which Sachs has termed amaurotic family idiocy.

Etiology.—There is a great tendency for the disease to appear in several members of a family, and hitherto all the recorded cases about the nature of which there is no doubt have been children of Jewish parents. The children, apparently healthy when first born, develop symptoms within the first few months.

Different views are held concerning the probability of a congenital or an acquired origin of the disease: the family characteristics certainly point to a congenital origin, and the idea that abnormalities of the mother's milk may be a cause appears to have been disproved by Sachs, who caused several of his patients to be reared by wet-nurses. Gordon Holmes and Poynton, after careful consideration of cases which they have examined, think that the disease is due neither to arrested development nor to bacterial toxins, but depends upon "some inherent bio-chemical property of the protoplasm of the cells" which brings about the degenerative processes.

Pathology.—There is a widespread degeneration of the cellular elements of the brain cortex,

with various degrees of sclerosis. Changes have also been noted in the basal ganglia and grey matter of the cord. Mott* concludes that the essential feature is failure in the elaboration of the nucleo-proteids of the neurones as evidenced histologically by the disappearance of the Nissl granules.

Symptoms.—The symptoms usually appear in the first few months of life—mostly between the third and sixth. The child becomes weak and wasted, and there is soon an obvious paralysis, which affects most of the body, and which may at first be of flaccid, and later on of spastic, type. At the same time the power of vision fails, and the mental faculties rapidly deteriorate.

The wasting and weakness progress, and *death* usually takes place in a few months.

The condition of the *reflexes* varies with the spastic and flaccid nature of the paralysis.

The *ocular* symptoms, first described by Waren Tay, are very characteristic of the disease, for, in addition to optic atrophy, there is, in the region of the macula lutea, a whitish patch, in the middle of which is a round spot of cherry-red colour. The appearance of this is similar to that occurring after embolism of the central artery of the retina (Plate XIII.).

Prognosis and treatment.—Up to the present, no methods for retarding the course of the degeneration have been discovered.

HYDROCEPHALUS

Hydrocephalus is characterised by the accumulation of serous exudation within the skull.

External hydrocephalus is the term applied to the collection of fluid between the dura mater and the brain, but the symptoms to which it gives rise are usually masked by the more important changes on which it depends.

* *Archives of Neurology*, Vol. iii., 1907.



PLATE XIII.—Fundus of the Eye, showing the Characteristic Appearance in Amaurotic Family Idiocy.

(From a Case under the care of Mr. Ilbert Hancock.)

Acute hydrocephalus occurs in association with various forms of meningitis.

Chronic hydrocephalus may be due to some congenital condition, or it may arise after birth as the result of meningeal inflammation or tumours. In many cases the chief factor seems to be obliteration

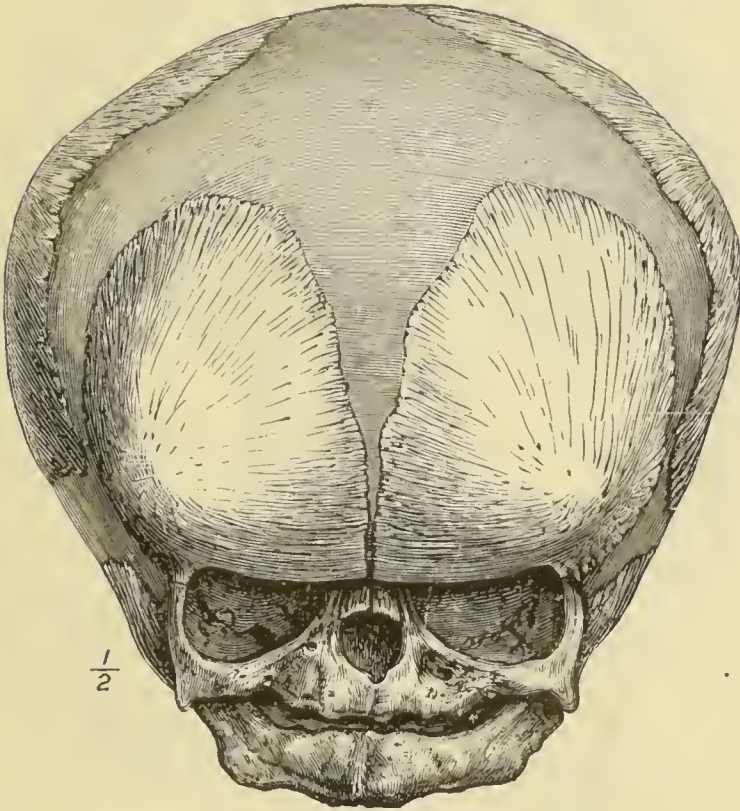


Fig. 70.—Hydrocephalic skull, showing Wormian bones.
(Museum, Middlesex Hospital. Bland-Sutton.)

tion of the foramen of Majendie and other passages through which the fluid normally circulates, added to which there may be an excessive secretion of the fluid.

The distension of the ventricles usually causes alterations in the brain, leading to mental impairment, but in some instances the intellect has remained good. Various degrees of diplegia may also be present.

In the hydrocephalic skull the bones are separated and the fontanelles distended. The frontal and parietal bones bulge outwards and overhang the eyes, the eyeballs being somewhat

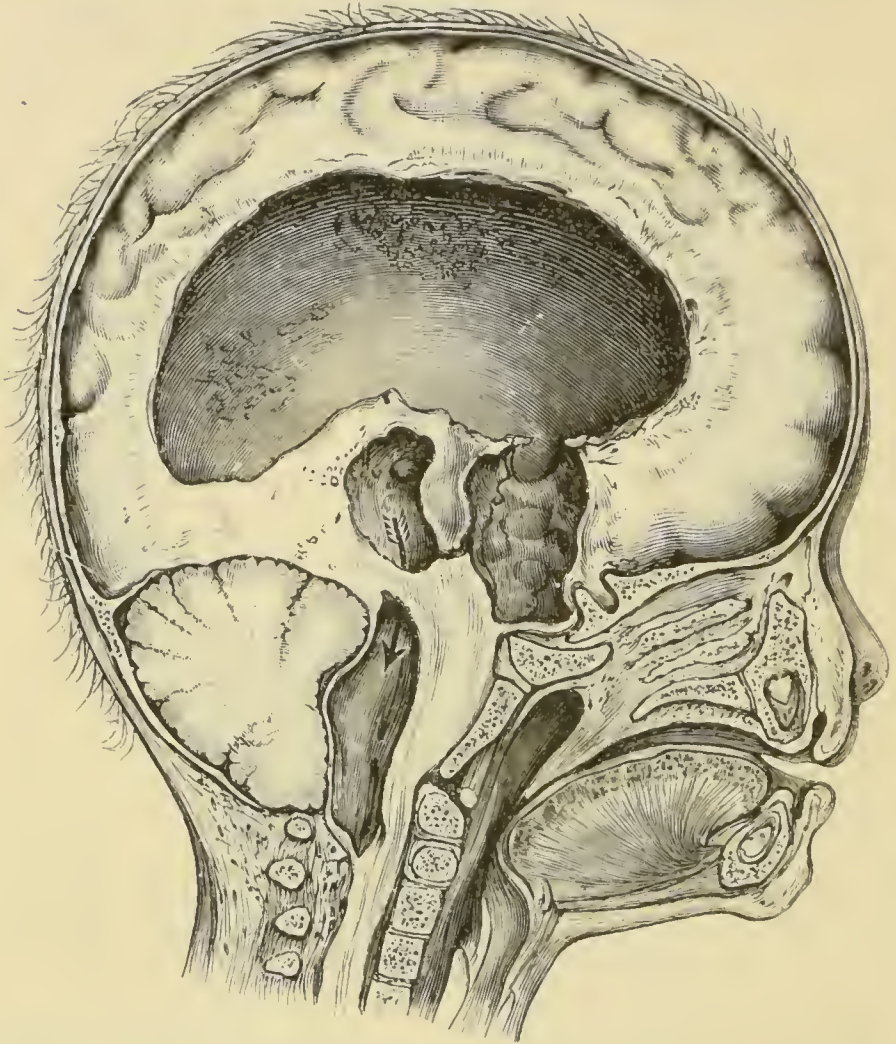


Fig. 71.—Sagittal section of a hydrocephalic skull from a child, with the brain *in situ*. The head of the arrow is in the growth, and its feathers in the third ventricle. The infundibulum is widely dilated. (Museum, Middlesex Hospital. Bland-Sutton.)

, depressed by the pressure of the orbital plates. The bones are sometimes softened, and Wormian bones occupy the parietal sutures. Distinction must be made between the hydrocephalic head and the square, flatter-topped head of rickets.

Prognosis.—In most cases of hydrocephalus the intellect is impaired, and the child usually dies young.

Treatment.—The most rational method of treatment is that of draining the fluid away, but hitherto the various means adopted have not met with much success.

have —

CHAPTER XXX

ACUTE ENCEPHALITIS ; CEREBRAL ABSCESS

ACUTE ENCEPHALITIS

SOME generalised inflammation of the surface of the brain probably always occurs in association with meningitis, and to a more limited extent after injuries such as fractures and punctured wounds of the skull. It may also be associated with areas of softening due to embolism or thrombosis.

A more localised form of inflammation, to which the name polioencephalitis has been given, is thought to correspond to acute anterior poliomyelitis of the cord, and is characterised by an acute degeneration of groups of cells in the cerebral cortex and of the nuclei of the cranial nerves. The resulting palsies, which may be of hemiplegic or cranial nerve distribution, are alluded to under the head of Cerebral Palsies of Children (Chapter XXIX.).

CEREBRAL ABSCESS (SUPPURATIVE ENCEPHALITIS)

Etiology.—The source of the infection in the case of a cerebral abscess may be in the skull or in some distant part of the body. Chronic suppuration of the middle ear is by far the most common cause, but infection may be carried equally well from other and neighbouring parts, and St. Clair Thomson has recently shown that diseases of the nasal sinuses are more important in this respect than has hitherto generally been supposed.

Necrosis of any other bones of the skull, arising

from injury, syphilis, or other diseases, may be followed by an abscess.

From more distant parts the infection is particularly apt to follow empyemata, bronchiectasis, and pulmonary abscess.

At other times the cerebral suppuration is but part of a generalised pyæmia, in which cases the abscesses are often multiple.

The posterior parts of the temporo-sphenoidal lobes, and the cerebellum, from their proximity to the bones of the ear, are the areas in which abscesses most commonly occur; less often the occipital and parietal lobes are infected. When the infective matter is brought from distant parts, the left side of the brain is rather more likely to be attacked than the right.

Pathology.—When the abscess follows diseased bone, the dura mater over the necrosed area is usually thickened, but the superficial part of the cortex generally appears healthy to the naked eye, and at a variable distance beneath its surface is situated the collection of pus. The channel by which the infection is conveyed to the brain is probably not always the same; sometimes it is by lymphatics, and sometimes by veins.

If the abscess has existed for some time, it is likely to be enclosed by a definite capsule, the result of organised products of inflammation in the neuroglial tissue; often, however, there is no such limit, and a large area of brain tissue may be permeated by pus, like water in a sponge.

The colour of the pus is usually a greenish hue, and the odour is generally foul. Streptococci and staphylococci can often be found, as also, occasionally, may tubercle bacilli and other specific micro-organisms.

Symptoms.—The symptoms of an abscess, as might be expected, resemble to some extent those of tumour, and they may be divided in a similar fashion into general and local—(p. 286).

Sometimes an abscess may exist for an indefinite time without producing any symptoms at all; and in chronic cases, *latent*, *acute*, and *terminal* stages may be well defined. In many there is a "latent period" of some length. When symptoms arise, headache, vomiting, and optic neuritis may all be present; but, compared with tumours, the pressure produced by an abscess is not often so great, and thus optic neuritis is not so constantly found, nor is it usually so intense, and for the same reason the vomiting and headache may be less troublesome. On the other hand, mental dulness is generally more pronounced than in the case of tumours. The slow pulse is present in both. In the terminal stages there may be signs of septic absorption which do not exist in most cases of tumour, *e.g.*, a quickened pulse and an irregular temperature, with occasional rigors; and in the last stages of the disorder there may be delirium and coma. The onset of these more general symptoms is suggestive of the coincidence of meningitis.

In many cases the septic symptoms are rendered more intense by absorption of septic matter from other regions besides that of the actual abscess.

Left to itself, an abscess which is giving rise to acute symptoms will cause death, either by direct pressure on vital parts, or after rupture into one of the ventricles, or by infection of the membranes and the supervention of symptoms of general meningitis.

The symptoms upon which the diagnosis of the position of the abscess depends are those which are discussed in dealing with the localisation of tumours (p. 289). In the *temporo-sphenoidal lobe* there will be the likelihood of sensory aphasia, or, if the abscess is in the fore part of the lobe, there may be, as in the instance narrated on page 301, a disturbance of the sense of smell. If the pressure is considerable the motor fibres may

suffer, and there will be signs of hemiplegia on the opposite side of the body.

When the pus is in the lateral lobe of the *cerebellum*, vertigo, nystagmus, paresis of the external rectus, with conjugate or skew deviation of the eyes, are among the prominent localising symptoms. One of the greatest aids in the diagnosis of the site of the abscess is the position of the primary suppuration, to which the abscess is secondary; the two are nearly always in close proximity, and in the majority of the cases the primary seat of disease is connected with the ear.

Diagnosis.—The principal local conditions from which abscess has to be distinguished are *meningitis*, suppuration in the mastoid cells, and thrombosis of cerebral sinuses; the difficulty in discriminating between these several conditions is increased by the fact that they may all be present at the same time.

If the disease is confined to the mastoid processes the pain, redness, tenderness, and other symptoms will be unaccompanied by any definite signs of implication of the brain. There will not be the mental dulness, vomiting, or optic neuritis, nor will there be any sign of pressure on any particular set of fibres. Moreover, the temperature will usually be raised instead of being subnormal, and rigors are common.

It is thus more often by a process of exclusion than otherwise that the diagnosis is made. In the same manner, careful consideration has to be given to the possibility of an abscess complicating sinus thrombosis, the symptoms of which are detailed in Chapter XXXIII.

In meningitis the signs are more generalised, and the cranial nerves are more likely to suffer. The patient tends to assume a curled-up position, is irritable, shuns the light, and objects to being touched; convulsions may also be present. The diagnosis may be aided by performing lumbar

puncture, for the cerebro-spinal fluid is liable to contain micro-organisms in the case of meningitis but not in abscess. Leucocytosis of the blood may be present in both cases.

A change in the percussion note over the side of the head in which the abscess is situated has sometimes been observed. Tenderness over the skull in the vicinity of the abscess is another sign that has occasionally been noted.

Prognosis and treatment.—The prognosis in the case of a patient with a cerebral abscess must always be doubtful. When there are several abscesses which are merely a part of some general pyæmic condition, the outlook is hopeless; but when there is only one abscess, the final result depends upon the possibility of opening and efficiently draining it.

If this can be accomplished, as is, fortunately, often possible when the abscess arises from disease of the middle ear and the mastoid, the chances of recovery are, on the whole, fairly good. It is obvious that no medicinal treatment can give relief, and therefore, when once the diagnosis is established there should be no time lost in resorting to surgical measures.

Infective Granulomata. } Syphilis.
Actinomycosis.

Carcinomata.

Sarcomata.

Gliomata.

benign Tumours

Cysts. { Simple.
Parasitic.

CHAPTER XXXI

INTRACRANIAL TUMOURS

Varieties.—The growths that occur in the brain comprise infective granulomata, carcinomata, sarcomata, gliomata, benign tumours of various kinds, and cysts of simple and parasitic origin.

With the exception of the carcinomata, the growths tend to occur before rather than after middle life, and are frequently found in children. Of the infective granulomata, tuberculous and syphilitic growths are the most common; indeed, tuberculous deposits occur more often than any other form of brain tumours. They are often multiple, and show a predilection for the cerebellum; they are chiefly met with in the young, and may sometimes subside into quiescence.

Gummata nearly always arise from the cerebral membranes, and only rarely are found deep in the substance of the brain. The commonest positions in which to find them are the base and cortex of the cerebrum, the structures of the cerebellum being invaded comparatively seldom. Gummata are often multiple and accompanied by a certain degree of meningitis, which causes further damage to the nerve substance around.

Actinomycosis has occasionally been found within the skull.

Carcinomata generally occur in later life, and are always secondary to a growth elsewhere.

According to Handley,* the cerebellum shows a special liability to be attacked.

* "Cancer of the Breast," 1906.

Sarcomata are either primary or secondary, and may arise in any part of the brain.

Endotheliomata grow from the membranes of the brain, and they differ considerably from one another in the intensity of their malignancy.

The psammomata, which contain earthy matter and cholesterin, belong to this last class.

Gliomata originate in the neuroglia. They infiltrate rather than compress the brain tissue. Owing to their close resemblance to the cerebral substance their boundaries are difficult to define, and their general appearance is often one of hypertrophy of a part of the brain. These tumours are usually single, and do not give rise to secondary deposits; they are, however, prone to undergo degeneration, and occasionally they become quiescent.

The *benign tumours* are of comparatively rare occurrence.

Neurofibromata occasionally grow from the sheaths of the cranial nerves. *Adenomata* and *cystic degenerations* may arise in connection with the pituitary body.

Cholesteatomata, lipomata, and angiomata occur occasionally, and sometimes an osteoma grows inwards and compresses the brain.

Parasitic cysts occur in connection with the *Tania echinococcus* and *Cysticercus cellulosæ*, and simple cysts are also sometimes found.

An intracranial aneurysm may give rise to symptoms of a tumour, according to its size and position.

Symptoms.—There are certain symptoms which may be associated with any cerebral tumour, irrespectively of its nature or situation, while others are peculiar—or accidental, so to speak—to the position in which the tumour happens to be placed. Thus the symptoms may be grouped under the headings of general and local.

The general symptoms are mainly the result

of increased intracranial pressure, and they include headache, vertigo, vomiting, optic neuritis, slow pulse, subnormal temperature, with possibly a general convulsion, and some mental failure.

It is hardly necessary to say that frequently not all of these are present in any given case, and that the absence of one or more of them by no means necessarily invalidates the diagnosis.

Headache.—Headache is one of the most constant symptoms of cerebral tumours. After it has once set in it seldom disappears, but its intensity is nevertheless liable to considerable fluctuations. The usual history is that of a dull ache with agonising exacerbations. When the growth is due to syphilis the exacerbations are most notable during the night, and there is often some localised tenderness, which can seldom be satisfactorily demonstrated in association with other kinds of tumours.

With the possible exception of the syphilitic cases, the position of the headache cannot in itself be relied upon to any extent as a guide to the position of the tumour, though, in conjunction with other symptoms, the seat of the pain may be strongly suggestive.

In cerebellar tumours, for example, the pain is often felt most intensely in the occipital region, and, if the other signs are in keeping, this localisation of pain may be given a certain value; but it sometimes happens that the headache of tumours in the posterior part of the brain is chiefly frontal in distribution, and, conversely, that of frontal tumours may be mainly felt at the back of the head.

Vomiting.—Vomiting is a common symptom. Sometimes it is preceded by nausea, while at other times the contents of the stomach are suddenly brought up without any warning. It may occur without any definite relation to taking food. Vomiting is liable to be very pronounced in

Several Symptoms.

Headache.

Vertigo.

Vomiting.

Optic neuritis.

Slow pulse.

Sub. Temp.

Gen. Conv.

Mental Failure.

tumours situated beneath the tentorium, where their growth is confined and causes a rapid rise of pressure.

Optic neuritis.—Of all the signs of cerebral tumour, optic neuritis is probably the most important. It occurs in a large percentage of cases, and, like the vomiting, is likely to be very intense, with subtentorial growths. The neuritis is sometimes more advanced on one side than the other, and such inequality has a certain degree of value in localisation by suggesting that the growth is in that half of the brain which corresponds to the inflammation of longer standing. Too much dependence must not, however, be placed upon it.*

It may also be said, roughly speaking, that the rapidity of the neuritis tends to be somewhat proportionate to the rate of growth of the tumour, though there must necessarily be many exceptions to this statement.

Temporary attacks of dimness or loss of vision, which are usually accompanied by exacerbations of headache and vertigo, are occasionally met with during the course of cerebral tumours, more especially of those of the cerebellum, and are regarded as being due to a temporary strangulation of the optic nerve or to pressure of the infundibulum upon the chiasma (Leslie Paton).

The slow pulse and the low temperature are both variable symptoms, and are produced by the increased intracranial pressure.

General convulsions, though important when they occur as an early symptom, are distinctly uncommon, but, unless their possible connection with tumours be remembered, they may easily be diagnosed as "epilepsy" until suspicions are aroused by the appearance of other signs. The greatest safeguard against overlooking a tumour

* Out of 50 cases analysed by C. E. Beevor, in 19 the neuritis was more intense on the side of the lesion, in 11 it was more marked on the side opposite the lesion, and in 13 it was equal on the two sides. In the remaining 7 there was no neuritis.





PLATE XIV.—Diagram of the Convolutions of the Cerebral Cortex.

consists in systematically examining the optic discs.

Mental changes sometimes occur quite apart from the disturbance of any special part of the brain, and are then to be attributed chiefly to the rise of pressure within the skull.

LOCALISATION OF CEREBRAL TUMOURS

The ability to localise with accuracy the position of cerebral tumours depends first upon a knowledge of the anatomical seat of those functions of the brain which are disturbed. Hence it is only possible to localise tumours accurately when they are situated in certain regions of the brain, for when they are in parts whose functions are unknown—the so-called “silent areas”—the general symptoms are accompanied by no special local disturbances by which the position of the tumour can be defined.

Another source of difficulty lies in separating the symptoms produced in the immediate vicinity of the tumour from those which arise some distance off as the result of indirect pressure effects.

The value of local symptoms also varies according to the date of their appearance. They are most reliable when their advent coincides with or shortly follows the appearance of the general symptoms, for when local symptoms appear only a long time after the onset of general symptoms, the chances are many, as shown by Collier, that they may not then point to the primary position of the growth, but rather represent the results of such secondary changes as hydrocephalus, meningitis, vascular lesions, and metastatic deposits; and, further, as the same author has also shown, the long-continued absence of local signs, when the general signs are well marked, is in itself of importance in suggesting that the tumour is situated above rather than below the tentorium.

since in the latter case localising symptoms generally appear early.

Assuming that the signs of a tumour are present, the various effects, according to the position of the growth, may now be considered.

I.—TUMOURS INTERFERING WITH THE MOTOR TRACTS

When the presence of a tumour is associated with loss of voluntary movements we may assume that the fibres which conduct the motor impulses are interrupted at some point in their course between the motor area of the cerebral cortex and the decussation at the medulla, and the different effects may be considered according as the growth is situated in (a) the motor part of the cortex, (b) the region of the internal capsule, (c) the optic thalamus, (d) the crus cerebri, or (e) the pons Varoli.

(a) **Tumours of the motor area of the cortex.**—The extent of the motor area of the cortex is seen from Fig. 72, which depicts the arrangement found by Sherrington and Grünbaum in the highest apes, and which presumably closely conforms to that of the human brain. It will be noticed that there are two important modifications of the plan which was formerly considered correct, viz. :

(1) The whole of the motor area is situated in front of the fissure of Rolando and is confined to the ascending frontal convolution.

(2) The representation for movements of the trunk is placed between those for the arm and leg, instead of being situated on the margin of the great longitudinal fissure.

Now, if a tumour be situated in this motor area, it is obvious that, at first, it will be likely to affect only a small portion of the convolution, and, as a general rule, the weakness is for a time confined to one limb or to the face, *i.e.*, there is a *monoplegia*, usually of slow development, which later on extends to a *hemiplegia* as the growing edges of

the tumour spread into the neighbouring areas. At the same time that the growth is reducing the power of movement, it is also irritating the motor-cells in its immediate vicinity. The result of this irritation is seen in convulsions, which begin in a

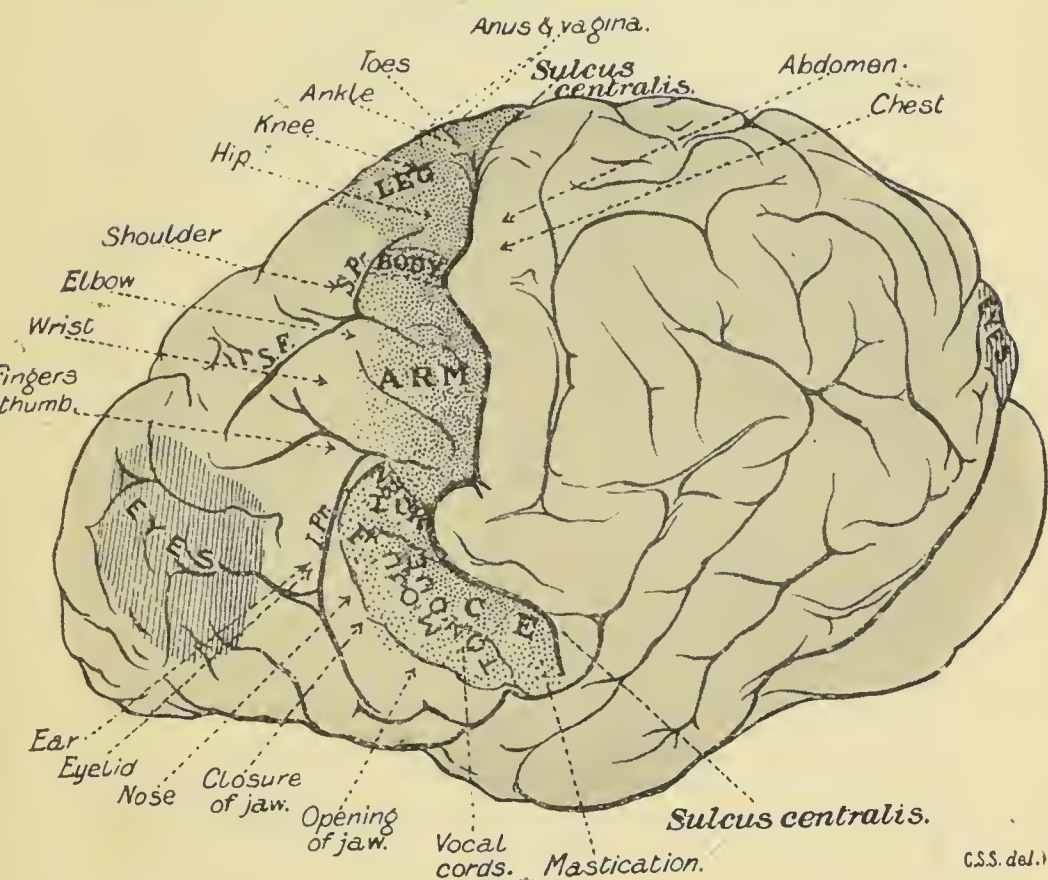


Fig. 72.—Diagram showing the situation of the motor areas in the cortex. (Sherrington and Grünbaum.)

group of muscles corresponding to the irritated area of the cortex, and subsequently spread with some deliberation to other parts of the limb, when they may either subside without loss of consciousness or develop into a general convulsion. The significance of these localised convulsions as the indicators of the particular area of cortex that is diseased was first pointed out by Dr. Hughlings

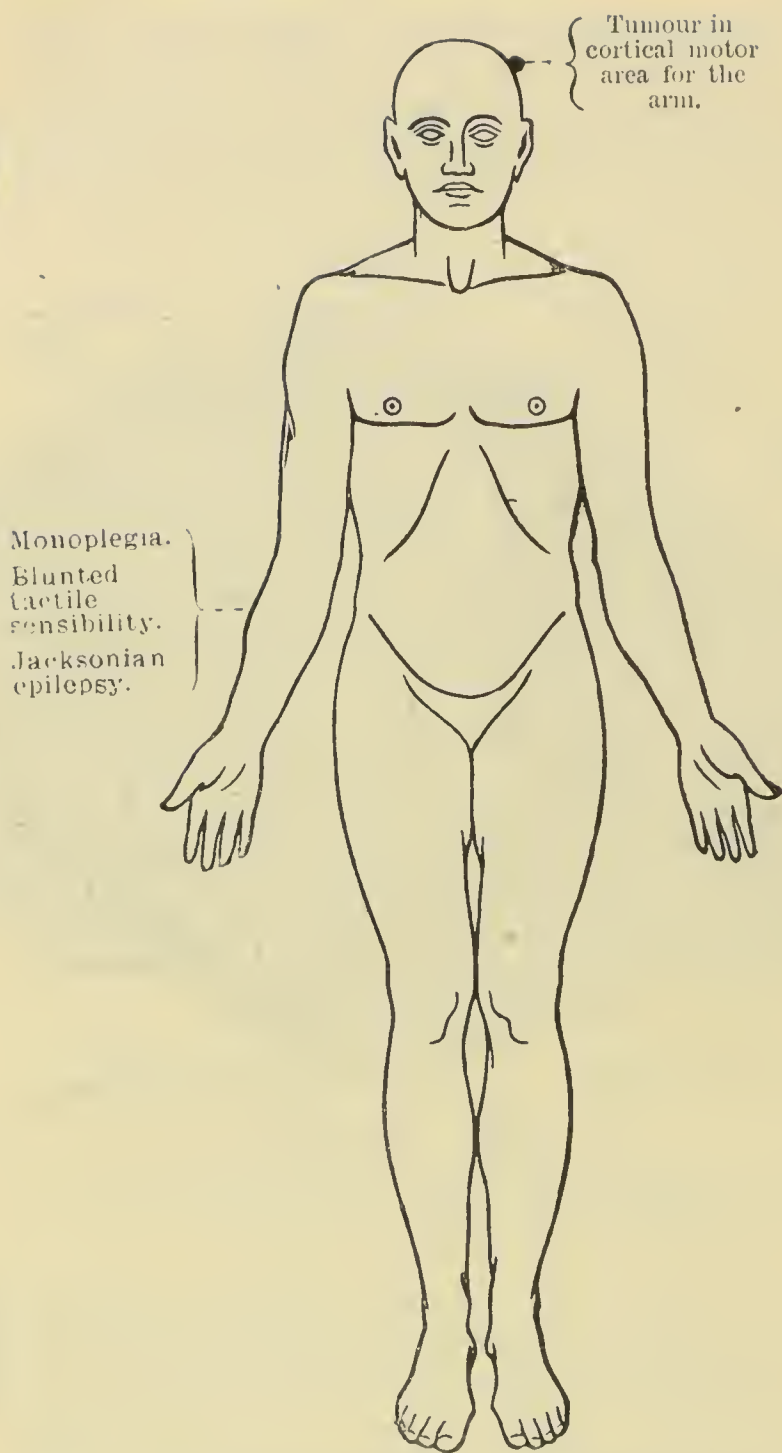


Fig. 73.—Diagram to summarise the principal symptoms produced by a tumour in the cortical motor area for the arm.

Jackson, from which they are known as Jacksonian epilepsy.

Localised convulsions are not in themselves pathognomonic of the presence of gross cerebral lesions, for they are frequently met with in the course of ordinary epilepsy. Their significance is the same in both cases, inasmuch as they indicate the part of the brain in which the epileptic discharge begins, but, where the general signs point to the presence of a gross lesion, their occurrence is of the greatest importance in enabling its position to be determined. Lastly, in addition to loss of power, a tumour of the motor cortex is also generally accompanied by some loss of sensation (probably due to pressure on the ascending parietal lobe), and, as Sir Victor Horsley has shown, this loss is chiefly concerned with the tactile sense. The patient does not feel so well as he should, and he is especially unable to tell exactly what point is touched. The extent of this anæsthesia corresponds roughly to that of the paralysis. To sum up, the main signs upon which reliance can be placed in the localisation of a tumour in the area of the motor cortex are monoplegia, Jacksonian epilepsy, and blunted tactile sensation.

(b) **Tumours in the region of the internal capsule.**—At the internal capsule the motor fibres are packed closely together, and a tumour pressing upon them is most likely to impair the functions of all, and so to cause a hemiplegia of slow onset. At the same time there may be a certain amount of hemianæsthesia, since the sensory fibres occupy the posterior third of the posterior limb of the capsule, but in slow-growing tumours this is often not noticeable. The fibres of vision from the occipital lobes may also be interrupted as they pass across the hindmost part of the internal capsule, in which case there will be hemianopia. There will be no Jacksonian

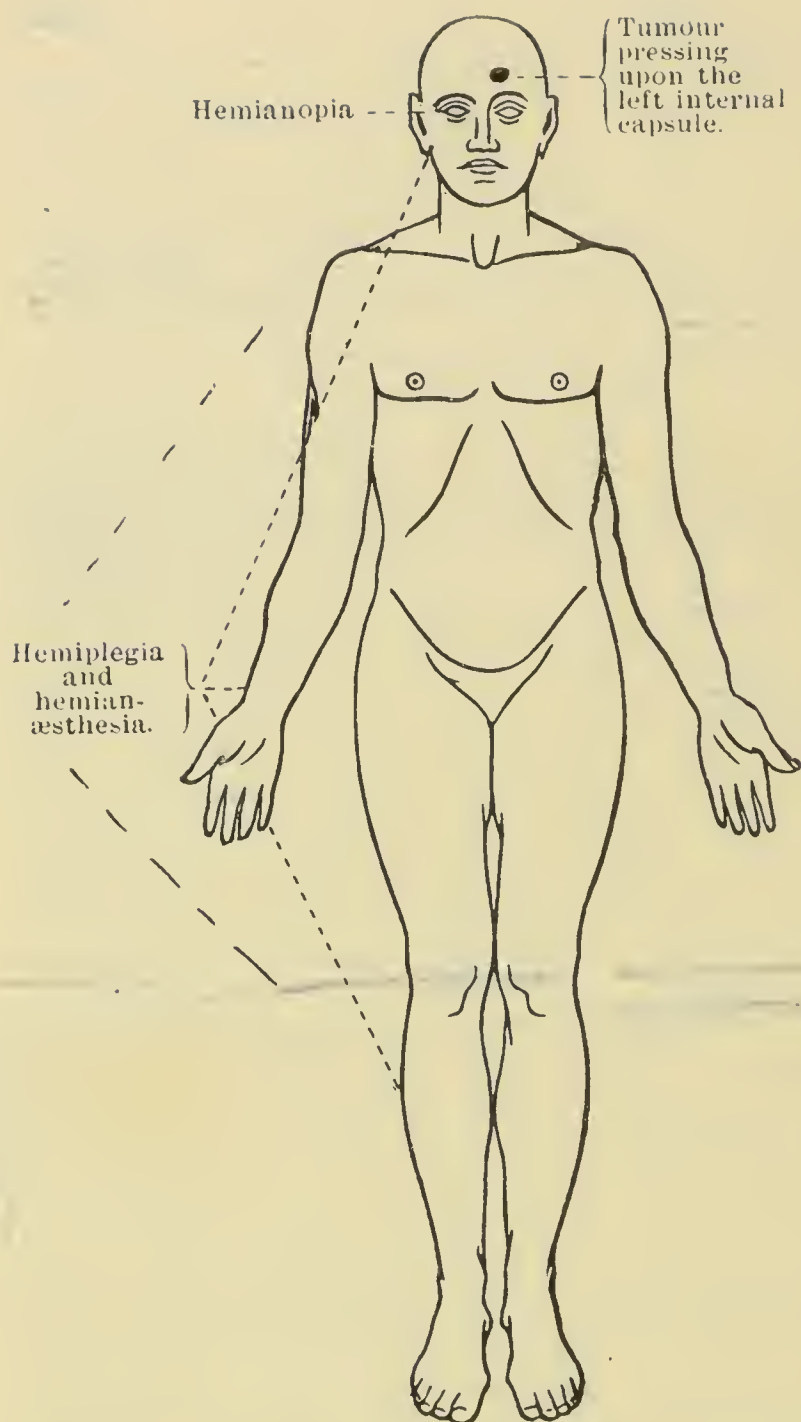


Fig. 74.—Diagram to summaries the principal symptoms produced by a tumour pressing on the internal capsule.

convulsions, though there may occasionally be a generalised fit due to the effects of general pressure on the cortex.

The main localising symptoms of a tumour pressing on the internal capsule are, therefore, hemiplegia, hemianæsthesia, and hemianopia.

(c) **Tumours of the optic thalamus.**—It is often very difficult to locate tumours of the optic thalamus. Most of the symptoms are due to pressure on the internal capsule, and therefore consist of hemiplegia, hemianæsthesia, and hemianopia. Loss of control over the emotional movements, the early appearance of anæsthesia and athetoid movements, are some of the signs that are most likely to occur.

(d) **Tumours of the crus cerebri.**—There will be the same tendency for a tumour of the crus to give rise to hemiplegia and hemianæsthesia of the opposite side as there is in the case of a tumour situated in the internal capsule, with the probable addition of paresis of certain cranial nerves, more especially of the third, from pressure on its nucleus or on the fibres springing directly from it. This gives rise to a "crossed" paralysis, i.e., the face, arm, and leg are paralysed on the one side (i.e., the side opposite the tumour), and the ocular muscles supplied by the third nerve are paralysed on the other side (i.e., on the same side as the tumour).

In addition, other cranial nerves, *e.g.*, the fourth, fifth, and sixth, may be pressed upon, if the tumour is a large one.

Tremors have also been observed in these cases.

Thus the main diagnostic points of a tumour in the crus cerebri are "crossed paralysis" (i.e., loss of power in the face, arm, and leg on the side opposite to the tumour, and paralysis of ocular muscles, and possibly facial muscles, on the same side), hemianæsthesia, and tremors.

(e) **Tumours in the pons.**—Tumours situated



Fig. 75. — Diagrammatic representation of "crossed" paralysis in which the face is affected on one side and the arm and leg on the other.

in the pons tend to cause symptoms somewhat similar to those of the crus, inasmuch as they give rise to hemiplegia and hemianæsthesia of the opposite side and also to a "crossed paralysis" (Figs. 75 and 76). In this instance it is the nucleus of the facial nerve that usually suffers on the same side as the tumour, but the oculo-motor (third), the abducent (sixth), and the auditory (eighth) may also be affected. It is also in a similar manner possible for lesions of the pons to cause "crossed hemianæsthesia," by damaging the fibres of the trigeminal nerve going to the one side, and those for the arm, trunk, and leg on the opposite side. There may be conjugate deviation of the eyes away from the side of the tumour. Optic neuritis is often comparatively late in making its appearance. The main points to be relied on are hemiplegia and hemianæsthesia on the opposite side, with facial paralysis of the peripheral type, deafness, and ocular palsies on the same side as the tumour.

In the *medulla* the motor fibres of the two sides are so close together that a tumour in their immediate neighbourhood is liable to cause bilateral paralysis, though in all probability the loss of power will be more marked on one side than the other.

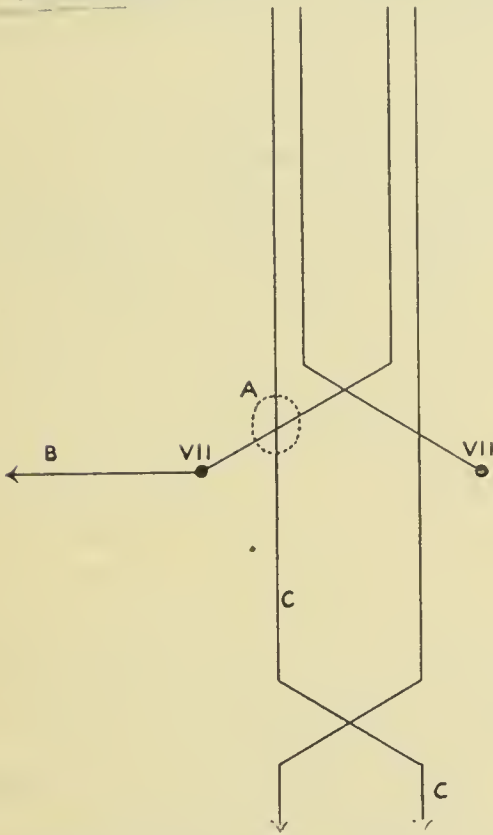


Fig. 76.—Diagram to show the mechanism of crossed paralysis. The lesion at "A" paralyzes the facial nerve "B" on the one side and the fibres for the arm and leg "C" on the other.

In addition to this, there is every likelihood of the existence of symptoms due to pressure on the bulbar nuclei, such as difficulty in speaking and swallowing, and weakness of the tongue. Glycosuria may also be found.

II.—TUMOURS SITUATED IN PARTS OF THE CEREBRAL CORTEX OTHER THAN THE MOTOR AREA

Tumours of the frontal lobes.—Tumours of the frontal lobes, whether cortical or subcortical,

are admittedly difficult to diagnose, since they may attain to a large size and yet give rise to no localising symptoms.

The functions of the frontal lobes are regarded as being mainly connected with the intellect, and hence mental disturbance, in conjunction with the absence of signs that the tumour is situated elsewhere, are the chief conditions upon which reliance must be placed. Mental disturbances are, however, often caused by tumours in other parts of the brain, although Mills, who has contributed largely to this important subject, considers that the conflict of evidence is more apparent than real, and that it would disappear if sufficient care were taken to analyse the symptoms and to exclude those which are of a more general character and obviously due to widespread irritation from a tumour. Mills believes that if every case of disease of the prefrontal lobes (and more especially of the left lobe) were carefully analysed, some mental symptoms of a special character in connection with the higher psychic activities would be recognised.

Grainger Stewart has observed that the superficial abdominal reflexes are often diminished or lost on the opposite side to the tumour, and that there is apt to be a fine, rapid, vibratory tremor of the limbs on the same side as the growth.

As the tumour grows, the signs of encroachment on the motor areas for the head and eyes, the limbs, or the motor centre for speech may give the clue to its position. It is generally believed that tumours of the posterior part of the third frontal convolution of the left side (Broca's convolution) cause motor aphasia, but in the light of Marie's recent observations (Chapter xxxv.) it is possible that this view will require revision.

Tumours of the corpora quadrigemina.
—In tumours of this region there is generally well-marked paralysis of the eye muscles, probably partly produced by pressure on the nuclei of the

third nerves. The reaction of the pupils both to light and to accommodation is often impaired, and nystagmus may occasionally be observed. There is also difficulty in maintaining the equilibrium and a tendency to fall in one direction.

Tumours of the corpus callosum.—It is but seldom that a growth beginning in the corpus callosum can be localised with any degree of certainty. As a rule the general symptoms develop slowly, and no localising signs occur until the tumour has invaded one or other hemisphere sufficiently far to injure the motor fibres. In some cases mental changes have been observed, suggesting a lesion of the frontal lobes.

Tumours of the parietal region.—A large number of sensory fibres impinge on the parietal lobes, and a disturbed function of these is the chief effect of a tumour of this region. The ascending parietal gyrus, which was formerly considered to be both motor and sensory in function, contains a large number of the fibres conveying tactile sensations, and Mills has pointed out that these fibres are arranged, like those of motor functions, in a definite order according to the areas which they represent. Each sensory area, thinks Mills, is probably on a level with the corresponding motor area, so that the representation of cutaneous sensibility of the face is probably situated in the lower third of the postcentral convolution, behind the seat of motor representation.

Muscular, thermal, and stereognostic sensibility are all to some degree represented in the cortex of the parietal region, and alterations in them all have been observed as the result of growths. The signs peculiar to a tumour of the parietal region are, therefore, modified sensations of touch, pain, temperature, and of form; but to these, others are usually added, though the encroachment of the tumour on the parts around. Thus, if it extends forwards, there will be motor

symptoms; if backwards, the functions of the angular gyrus and occipital lobe will be impaired; while if the disease spreads beneath the cortex, it may affect the motor fibres as they pass through the corona radiata. Convulsions associated with tumours of the parietal lobes are usually preceded by sensory warnings.

When the tumour encroaches on the angular gyrus there may be a concentric contraction of both fields of vision, but greatest on the side opposite the tumour (crossed amblyopia). Visual aphasia (word-blindness) may also occur from tumours of this region when situated on the left side. Fits beginning with visual warnings may also be associated with tumours of the occipital region.

Tumours of the occipital lobes.—Fits beginning with visual warnings may be the early signs of tumours of the occipital region of the cortex. If the tumour is in the cuneate lobe there will be hemianopia, i.e., loss of half-vision in the corresponding parts of the two fields (homonymous or simple hemianopia); but the value of this symptom is, of course, frequently lost through the general impairment of vision from optic neuritis. Occasionally only a quadrant of the usual field is lost (quadrantic hemianopia). If the angular gyrus is injured on the left side there may be word-blindness.

Tumours of the temporo-sphenoidal lobes.—Tumours of the temporo-sphenoidal lobes may cause deafness on the opposite side, and, if situated on the left side, there may also be sensory aphasia (word-deafness). Auditory warnings may occur before a fit, or hallucinations of hearing may be present. Extension of the tumours inwards may cause pressure on the motor and sensory fibres for the opposite limbs.

Tumours of the uncinate gyrus.—Tumours of the anterior part of the temporo-sphenoidal

lobe are sometimes accompanied by the "dreamy states," first described by Hughlings Jackson.

Sensations of smell may also accompany tumours in this region, a fact which supports the view that the cortical area concerned with olfactory sensations is that of the uncinate gyrus or its immediate neighbourhood.

One such case which the writer had an opportunity of observing was that of a young man under the care of Dr. J. Kingston Fowler,* at the Middlesex Hospital. Along with general signs of cerebral abscess there were frequent attacks of epileptic vertigo, which were preceded by a sense of an unpleasant smell. After death a large abscess was found in the fore part of the temporo-sphenoidal lobe.

Another important fact connected with this case was the excessive hunger and thirst from which the patient suffered, a condition which is occasionally found in connection with injuries and diseases of various parts of the brain, but more especially, it would appear, when the lesion is in the locality of the temporo-sphenoidal lobe, a point which has been brought out by Stephen Paget† from a survey of fourteen cases of the kind which he collected.

Thus sensations of smell, together with "dreamy mental states," and perhaps voracious hunger and thirst, are the signs which suggest that a tumour is situated in the neighbourhood of the uncinate convolution.

III.—TUMOURS AT THE BASE OF THE BRAIN

Tumours at the base of the brain are generally accompanied by early signs of palsy of cranial nerves, the situation of which is depicted in Plate xv.

In tumours of the pituitary body the optic

* See "*Post-mortem Records of the Middlesex Hospital*," 1895.

† *Transactions of the Clinical Society of London*, 1897.

chiasma is very apt to suffer, and if it be compressed at its centre a double temporal hemianopia will ensue, followed subsequently by more complete loss of vision as the pressure extends to the fibres which constitute the temporal portions of the optic nerves (*see* Fig. 8, p. 45). These symptoms are often the first complained of by patients suffering from *acromegaly*, a disease which is mainly characterised by enlargement of the bones, more especially those of the head, face, hands, and feet—changes which are also generally accompanied by curvature of the spine.

Tumours growing in the middle fossa generally paralyse the ocular and trigeminal nerves, while those of the posterior fossa affect the sixth nerves very early, and signs of pressure on the crus, pons, and cerebellum may appear.

Tumours at the angle of the pons and cerebellum.—Tumours growing at the angle of the pons and cerebellum are of special interest from the relative frequency of their occurrence and the possibility of their removal. Although this region of the brain is not exempt from tumours of every kind, the one that is especially prone to arise is of a modulated fibrous appearance, which not unfrequently attains the size of a walnut or a pigeon's egg.

These growths have been variously described as fibro-neuromata and sarcomata, and often appear to arise from the sheath of the auditory nerve. Bland-Sutton looks upon some of them as psammomata, and believes that they have originated from processes of the chorionic villi of the fourth ventricle. Sometimes they are bilateral.

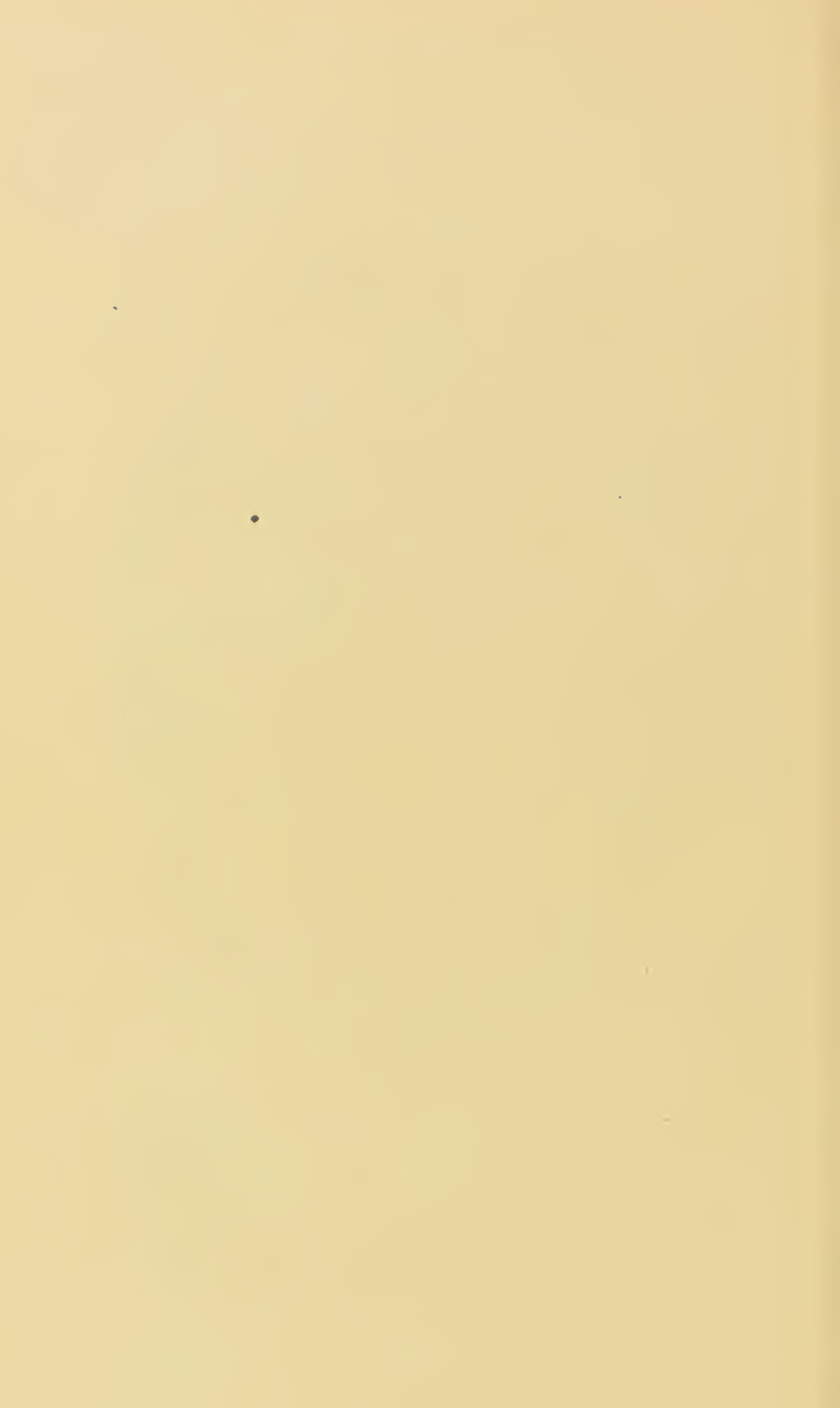
These tumours produce localising symptoms from pressure—

(1) On the cranial nerves in their immediate vicinity.

(2) On the cerebellum.



PLATE XV.—The Cranial Nerves at the Base of the Brain.
(From a Painting by Sir Charles Bell in the Middlesex Hospital
Medical School.)



(3) On the *pons* and *medulla*.

Reference to Plate xvi. will make these three lines of pressure clear.

1. *Pressure on the cranial nerves*.—The nerves most likely to suffer are the fifth, sixth, seventh, and eighth, and, as the tumour generally arises in close apposition to the auditory nerve, deafness is often one of the earliest symptoms.

The facial trunk is generally compressed at the same time, but it is apparently more capable of resisting pressure than the auditory nerve, so that the facial muscles often suffer to a surprisingly slight extent.

The sixth nerve is affected early through displacement of the parts, and weakness of the external rectus with its corresponding symptoms results.

The fifth nerve becomes compressed as the tumour extends forward, and, in consequence, pain is complained of over some part of the area of its distribution.

2. *Pressure on the cerebellum*.—As the tumour presses backwards on to one of the lateral lobes of the cerebellum it gives rise to all the symptoms of a tumour primarily situated in that position, viz.: nystagmus, ataxy, lurching in one or other direction, weakness of the arm and leg on the same side, and frequent attacks of giddiness—symptoms which are also discussed under the heading of Cerebellar Tumours.

3. *Pressure on the pons and medulla*.—The pons and medulla are often greatly displaced towards the opposite side, a point which is particularly well shown in the accompanying illustration (Plate xvi.).

The displacement tends to cause a weakness of the arm and leg of ordinary hemiplegic character on the *opposite* side, since the motor fibres to the limbs are compressed a little above the point at which they decussate. The face on

the opposite side to the tumour will usually be unaffected (the probability of some facial paralysis on the SAME side as the tumour has already been mentioned), since the fibres to the nucleus of the seventh nerve have already crossed and are usually beyond reach of the tumour. Considering the degree of the displacement that often occurs, one would expect the effects to be very great, but they are, in fact, often but slight, for, the growth being slow, the fibres are so gradually compressed that they are able to adjust themselves to the altered conditions with very little loss of function.

In the case illustrated there was no definite hemiplegia on the opposite side to the tumour, and it affords an excellent example of the power of preservation of function which nerve fibres possess when the pressure on them is only very slowly increased. The main points to be borne in mind for localising tumours in this situation are—

(1) Pressure on the auditory (chiefly), facial, abducens (sixth), and trigeminal (fifth) nerves on the same side as the tumour.

(2) Symptoms of disease of the lateral lobe of the cerebellum, also on the same side.

(3) Weakness of arm and leg, with increased deep reflexes and extensor type of plantar reflex on the opposite side.

Since these tumours usually press upon three different sets of structures, namely, cranial nerves, cerebellum, pons and medulla, it is obvious that difficulty must sometimes be experienced in deciding which of the three parts is the primary site of the growth. In other words, is the growth situated in the angle between the pons and cerebellum, in the cerebellum, or in the pons?

Early signs of implication of the cranial nerves—as, for instance, when deafness, along with weakness of the facial muscles and noises in the ear, makes its appearance slowly and gradually,

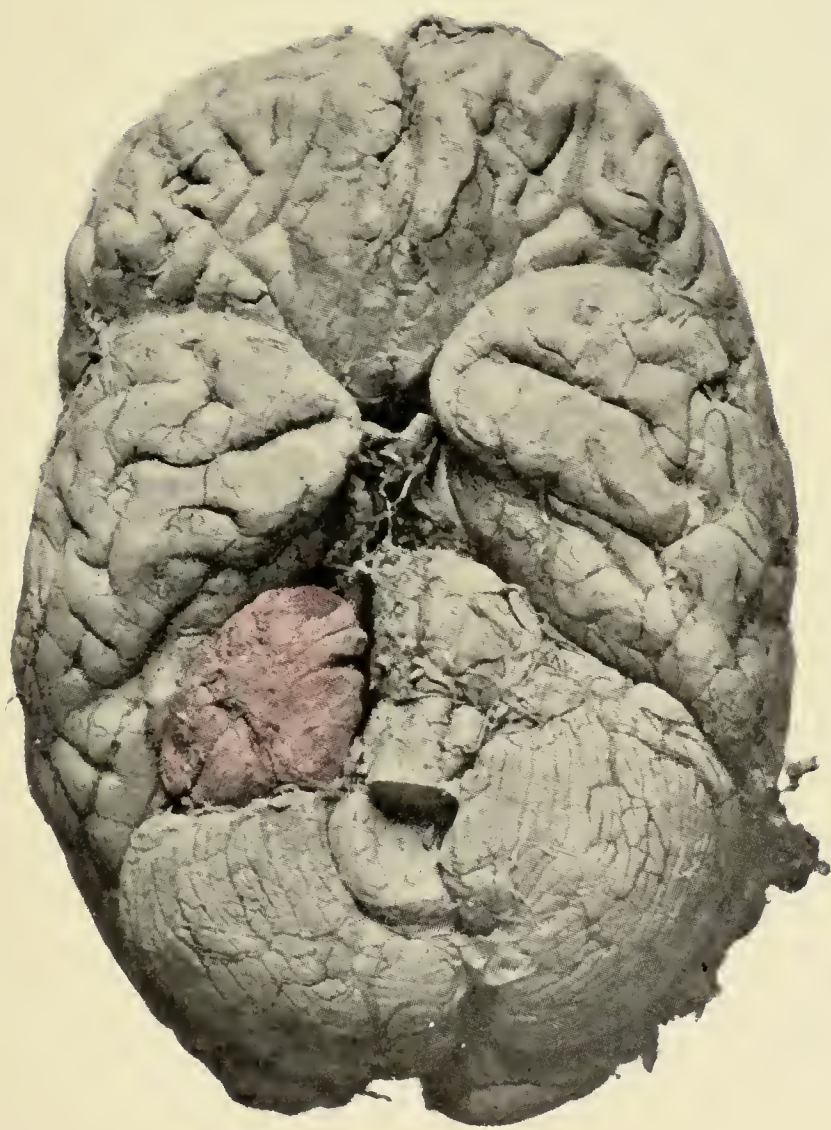


PLATE XVI.—Tumour situated at the Angle of the Pons and Cerebellum.

before anything else is noticed are strong evidence that the tumour is in the pontine-cerebellar angle. In cases of primary intracerebellar growth, these cranial nerves, with the exception of the sixth, would not be likely to suffer till other symptoms were well developed. Grainger Stewart and Gordon Holmes have observed that, in the vertigo accompanying tumours of the lateral lobe of the cerebellum, the direction in which the patient feels himself to be rotating is away from the side of the lesion; while in cases of extra-cerebellar tumours the sensation of turning is towards the side of the lesion.* The chief effects of tumours of the pons have already been considered, and the prominence of "crossed paralysis," conjugate deviation of the eyes, and paresis (sometimes bilateral) will generally serve to distinguish them from the tumours now under consideration.

IV.—TUMOURS OF THE CEREBELLUM

In tumours of the cerebellum the general symptoms of an intracranial growth are, as a rule, exceptionally well marked.

Optic neuritis is more constantly met with in association with these tumours than with those of any other part of the brain. It comes on early, and the inflammatory process is apt to be more advanced in the eye of the same side as the growth. Headache is usually very intense and often referred to one or other side of the occipital region; there may also be rigidity of the neck muscles. Vertigo and vomiting are likewise generally prominent symptoms and appear at an early date.

The experiments of Risien Russell† and others have shown that the influence of one half of the cerebellum is chiefly exerted on the same side of

* *Brain*, Part cviii.

† *Phil. Trans. Royal Society*, 1894, Part ii.

the body, in contradistinction to that of the cerebrum, where each half mainly governs the opposite side; tumours and other diseases of the cerebellum therefore tend to produce symptoms on the same side of the body as the lesion.

The signs of a cerebellar tumour may be divided into two groups, according as they depend directly upon the disordered function of the cerebellum, or indirectly upon pressure on neighbouring parts.

1. **Direct effects of cerebellar lesions.**

Motor symptoms.—These are generally *weakness and want of tone in the limbs on the same side as the tumour.* This loss of power, often slight, on the same side as the lesion, and due directly to deficiency of cerebellar function, must not be confused with paresis on the opposite side, which may arise from the effects of pressure on the medulla or pons.

Incoordination, usually more prominent on the side of the lesion, is especially marked in the lower limbs, but is not usually increased when the eyes are closed. One effect of incoordination of the upper limbs which is sometimes seen is an inability to pronate and supinate the forearm rapidly.

The gait is of a reeling character, and the legs are kept wide apart; there is also a tendency to turn and fall to one side, usually, though not always, towards the side of the tumour.

Compensatory attitudes are sometimes assumed in attempting to neutralise the tendency to incline towards the diseased side. These are often excessive, and cause the patient to incline unduly towards the sound side.

Tremors are often present.

Tonic spasms, causing opisthotonos and rigidity of the whole body, have sometimes been noted, but they are uncommon. Occasionally there are attacks of jerky movements, which in one case

that the author had an opportunity of observing began in one arm and then spread to the opposite limb. Unless care be taken to inquire closely into the character of such attacks, a patient's description of the condition may suggest some similarity to a Jacksonian fit.

Position of head—It has been shown that in a certain number of cases the head is tilted so that the ear approximates to the shoulder on the side opposite the lesion, while the face is turned up to the side of the lesion.

Forced movements, in which the patient rotates round the long axis of his body, sometimes occur.

Ocular movements.—*Nystagmus* is very frequently present. Sometimes it is lateral, and brought out by fixing the eyes to one or other side; at other times it is associated with all the movements of the eyeball, and it may be of a rotatory kind. The movements are often of different character, according to whether the eyes are turned to the right or left, being short and sharp in the one instance, and longer and slower in the other. In such cases the more deliberate movements generally occur on the same side as the tumour.

Skew deviation, where one eye is turned downwards and inwards and the other upwards and outwards, is sometimes observed. In a case of cerebellar abscess recently under the care of the writer, both eyes were turned downwards and inwards.

Weakness of the external rectus, which is so frequent in cerebellar disease, is due to pressure on the sixth nerve, and reference is made to this in the paragraph dealing with the indirect results of these tumours (p. 308.)

Vertigo is generally a prominent symptom and occurs in paroxysms.

Reflexes.—The knee-jerks vary in different cases; if they are unequal, the more active, according

to experimental results, is likely to be on the side of the lesion (Risien Russell).

In tumours of the middle lobe there may be retraction of the head, arching of the back, and extension of the legs (cerebellar attitude of Hughlings Jackson), which position may be further intensified by the occurrence of tetanus-like seizures. There is generally a tendency to fall backwards.

2. **Indirect effects.**—Of the symptoms produced by pressure on the parts around, the most frequent is a paralysis of the sixth nerve on the same side as the lesion, which leads to weakness of the external rectus and subsequently to a tendency for the eyes to deviate from the affected side. Other cranial nerves, such as the auditory, facial, and trigeminal, may be injured, though, as a rule, much less severely in cerebellar tumours than in those which arise at the angle of cerebellum and pons.

Pressure on the pons may cause "crossed paralysis," *i.e.*, hemiplegic symptoms on the opposite side of the body, with ocular paralysis on the same side; while pressure on the medulla may produce pharyngeal and laryngeal symptoms. Attacks of dyspnoea are also apt to occur from pressure on the medulla, and are of serious import.

When the tumour grows slowly it may cause hydrocephalus, the symptoms of which may mask those produced directly by the primary disease.

Diagnosis of intracranial tumours.—

The diagnosis of cerebral tumour is made from the general signs of a growth, aided by the local ones, if present.

Mistakes are most likely to arise when headache and vomiting and perhaps optic neuritis are present from other causes, such as Bright's disease, severe anæmia, and chronic lead poisoning. Where

the symptoms are very obscure a case may even for a time be regarded as functional. Having decided that a cerebral tumour is present, it is necessary to try (1) to locate its position, (2) to infer its probable structure, and (3) to decide whether any attempt shall be made to remove it.

Prognosis and treatment.—The prognosis must depend upon the conclusions that are arrived at concerning (1) the nature of the tumour, (2) the probability of its removal by medical or operative measures, and (3) the possibility of the occurrence of spontaneous retrogression. A benign tumour is, other things being equal, obviously more hopeful than one that is malignant, and should the latter be a secondary deposit the outlook will in most cases naturally be hopeless. It is in syphilitic growths that most is to be expected from medicinal treatment, and when there is the least chance of the disease being of this nature a thorough course of mercurial inunction, together with full doses of potassium iodide taken internally, should be given. Tuberculous masses often show a tendency to retrogress under the influence of cod-liver oil, iron, good food, and fresh air.

With these exceptions no reliance can be placed on drugs, and, if no benefit be obtained from a thorough but brief trial of treatment by mercurials and iodides, the desirability of an attempt to remove the tumour must be earnestly considered. The decision must depend chiefly on the apparent accessibility of the growth and on the probable effects of its attempted removal.

Tumours of the frontal, parietal, occipital, and temporo-sphenoidal cortex can all be reached, as also those of the cerebellum and cerebello-pontine angle.

Where removal of the tumour, owing to its size or position, cannot be accomplished, an endeavour should be made to preserve the sight by

relieving the intracranial pressure, the importance of which is especially seen in cases where the growth becomes quiescent. Care must be taken, therefore, not to allow the changes in the retina to proceed too far before something is done, and during the period that medicinal treatment is being carried out careful observations must frequently be made on the condition of the optic discs and on the fields of vision, for any rapid contraction of the latter indicates the necessity of relieving the pressure without further delay. For this purpose a considerable piece of bone should be taken away and the dura mater excised. If no attempt is to be made to deal with the tumour, Sir Victor Horsley* recommends the basal temporal region of the right side as a suitable spot to trephine.

* *Brit. Med. Journ.*, Aug. 25, 1906.

CHAPTER XXXII

CEREBRAL HÆMORRHAGE ; CEREBRAL THROMBOSIS ; CEREBRAL EMBOLISM

CEREBRAL HÆMORRHAGE

Etiology.—Arterial degeneration accompanied by a rise of blood pressure are the main factors concerned in the production of cerebral hæmorrhage.

These two conditions are most often associated with *chronic interstitial nephritis*, and it is during the course of this disease that a large number of cases of hæmorrhage occur.

It naturally happens that the causes which lead to the final rupture of a vessel are often complex and difficult to analyse, since they include all those which tend to produce arterial degeneration, some of the principal of which are *alcoholic excess*, *chronic lead poisoning*, *syphilis*, and *senile degeneration*.

It is particularly important to remember the tendency shown by the blood pressure to rise during the later years of middle life, even when there are present no recognisable signs of renal disease, and no doubt, as Sir Clifford Allbutt has strongly insisted, a more general and timely recognition of this fact, combined with suitable treatment, would lead to many attacks of apoplexy being averted.

The obliterative endarteritis from syphilis more often leads to thrombosis than hæmorrhage,

as also does the degeneration which accompanies very old age.

The arteries of men break more often than those of women, and the period in which most cases occur is from 45 to 65 years of age. Sudden exertion may precipitate the attack, but in a considerable number of instances the artery breaks while the patient is asleep. Hæmorrhage sometimes takes place into a tumour of the brain, and it may also be associated with changes in the blood, as in leucocythæmia, pernicious anæmia, purpura, septicæmia, and scurvy, but the symptoms produced in these latter cases are seldom those of typical apoplexy.

Pathology. — The arteries in a case of cerebral hæmorrhage generally present the usual appearances of an advanced stage of arterio-sclerosis. Their walls are rigid and brittle, and small aneurysmal dilatations, the rupture of one of which is the cause of the hæmorrhage, can frequently be seen with the naked eye or a magnifying glass.

The appearance of the blood differs according to the length of time which has elapsed since the hæmorrhage occurred. At first there are all the appearances of freshly-extravasated blood, but, after a clot has formed, a gradual shrinkage takes place, and the last appearances may be those of a scar or a cyst with discoloured walls containing some clear or brownish fluid.

Symptoms. — Symptoms which depend on degenerated vessels and high blood-pressure, such as headache and giddiness, are often complained of by people whose arteries are likely to rupture, and sometimes more distinct warnings, such as a ptosis or squint of passing duration, are met with. Many of such signs are probably due to minute hæmorrhages, the tendency to which may often be seen by looking at the retina.

A hæmorrhage causes symptoms (1) by the rise

of intracranial pressure which it produces, and (2) by destroying the function of the nerve fibres in the particular locality in which it occurs.

When a hæmorrhage is quite small there may be symptoms of local injury without any disturbance of consciousness, and it is probably in this way that the temporary ptosis and squints sometimes preceding a more severe attack are produced.

On the other hand, consciousness may be considerably disturbed without any permanent local effect.

Classification of symptoms.—It is convenient to classify the symptoms into:

(1) General: common to all hæmorrhages of a certain size, irrespective of their situation.

(2) Local: depending on the position of the hæmorrhage.

(1) The chief *general symptoms* are: loss of consciousness, varying in degree from confusion to coma; stertorous breathing, which may be of the Cheyne-Stokes type; slow full pulse, subnormal temperature, and loss of power over the sphincters (this last depends on the depth of unconsciousness).

(2) The *local symptoms* necessarily vary with the position of the hæmorrhage, which may be anywhere in the brain, but the cerebral vessels which most often break are the branches of the lenticulo-striate artery, which come off directly from the middle cerebral and pierce the base of the brain to supply the neighbourhood of the lenticular nucleus. These vessels are in close proximity to the motor fibres as they pass through the internal capsule, and an effusion of blood of any size in this neighbourhood nearly always causes loss of power on the opposite side of the body (Plate xvii.).

The hæmorrhage is usually large enough also to cause loss of consciousness, and hence, in the

greater number of cases, the symptoms bear a close resemblance to one another.

As a *typical example*, it is therefore convenient to discuss the results of *hæmorrhage* and *hemiplegia* resulting from rupture of the *lenticulo-striate artery*.

Onset.—When the attack takes place in the daytime the patient feels giddy and, after perhaps reeling for a few steps, sinks in a dazed manner to the ground or on to a chair, and quickly loses consciousness. It is usually after this condition has existed for some little time that he is seen by the physician; the patient is then completely unconscious, breathing stertorously, and has a full, slow, bounding pulse and a turgid countenance. Perhaps the head and eyes are persistently turned to one or other side.

Diagnosis.—An attempt at this stage must first be made to determine the *cause* of the *coma*, but for a time it may be impossible to arrive at any definite conclusion.

The chief conditions besides cerebral hæmorrhage that suggest themselves in such a case are uræmia, diabetes, alcohol, opium or other narcotic, post-epileptic coma, and concussion.

In *uræmia* the attacks may for a time very closely resemble those of cerebral hæmorrhage, especially if, as sometimes happens, the power of movement on the two sides of the body is unequal. Moreover, cerebral hæmorrhage is likely to occur in the very people who are also liable to uræmia. In some cases, twitchings and convulsions, together with the history of the previous symptoms, afford some assistance, but in others the diagnosis must for a while remain in doubt.

Coma due to *alcohol* often gives rise to difficulty, and the danger lies in pronouncing a man to be drunk when he has been the subject of a hæmorrhage while drinking. The evidence of drink does not include the possibility of hæmorrhage.



PLATE XVII.—Section of Brain showing the Anatomy of the Internal Capsule.

A, Caudate Nucleus; B, Lenticular Nucleus;
C, Internal Capsule; D, Optic Thalamus.

and, when there is the least doubt, the only safe course is to keep the patient under careful control for a few hours.

The coma of *diabetes* generally comes on rather more gradually than that of hæmorrhage. The dyspnœa is generally that of the "air hunger" type, and lacks the deep stertor that accompanies hæmorrhage, nor is there any sign of unilateral paralysis. If a specimen of urine can be obtained the presence of sugar will make the diagnosis clear.

The signs of *opium* poisoning closely resemble those of hæmorrhage into the pons by the contracted pupils and deep coma. Fortunately there are generally some indications in the history which lead to the suspicion of poisoning.

The coma which follows an *epileptic fit* is sometimes very deep, but there is generally an account of the convulsion even if the history of a previous liability to fits be not obtainable.

In *concussion*, the difficulty of deciding whether there is a meningeal hæmorrhage may arise, for it very rarely happens that an intra-cerebral artery ruptures from the direct effects of a blow. When a meningeal artery bleeds, the signs are those of an increasing pressure on the brain, possibly accompanied by convulsions.

In the course of the examination of a case of supposed cerebral hæmorrhage, careful observation often shows signs of one-sided weakness. One cheek puffs out loosely, and the arm and leg on the same side when raised fall in a heavy, dead fashion, which, in spite of the loss of consciousness, obviously differs from that of the other side and justifies the conclusion that a one-sided cerebral lesion is the cause of the trouble.

The ground is cleared considerably when this conclusion can be arrived at, since a one-sided lesion coming on suddenly in a man who has up to that time been in ordinary health almost

certainly indicates a vascular lesion, and so the issue is narrowed to *hæmorrhage*, *thrombosis*, or *embolism*.

Embolism must be excluded by the absence of a cause, such as valvular disease of the heart, and moreover the deep loss of consciousness would at once weigh heavily against it, for although blocking of the middle cerebral artery or one of its branches will cause a good deal of mental confusion, especially if complicated with aphasia, there is nothing in its nature to produce the deep coma that accompanies the rapid rise of intracranial pressure consequent on the effusion of a quantity of blood.

In the diagnosis between *thrombosis* and *hæmorrhage*, often a most difficult matter, the main points to be considered are—

1. Loss of consciousness.—The existence of extensive paralysis without any definite loss of the senses is highly suggestive of *thrombosis*, for a sudden effusion which is large enough to cause hemiplegia is most likely also to raise the intracranial pressure sufficiently to cause loss of consciousness. Of course, an exception to this general rule may occur when a small hæmorrhage takes place somewhere exactly in the course of the motor fibres.

2. Onset of paralysis.—In the case of hæmorrhage the onset of paralysis is generally sudden and does not show signs of extension, for the damage is usually all done in a very few minutes. On the other hand, a paralysis which progresses over a period of some hours, especially if there is no loss of consciousness, is very characteristic of *thrombosis* in which the clot is extending from the original thrombus into neighbouring vessels. Before the onset of paralysis there may have been numbness, tingling, or a passing weakness in a limb, signifying that the blood supply was very deficient before the occlusion finally occurred.

3. *The vascular system.*—The association of an hypertrophied heart with degenerated arteries is in favour of hæmorrhage. High arterial tension also disposes to hæmorrhage, and further evidence may be obtained from signs of the existence of chronic interstitial nephritis.

4. *Age.*—In the very old, thrombosis is more likely to occur than hæmorrhage. In the young and in early middle life, say under forty, cerebral hæmorrhage is exceedingly rare, while thrombosis is comparatively frequent from syphilitic endarteritis.

Treatment during the early stage.—As soon as the diagnosis of cerebral hæmorrhage has been made it is necessary to proceed at once to treat the patient.

In rare instances, where the patient is seen at the moment the bleeding is taking place, prompt venesection and pressure on the carotid arteries might be of use; but, as a general rule, the time for these measures has gone before any opportunity of treating the patient arises. The best method then of trying to prevent further oozing is to keep the patient at absolute rest, and to calm down the circulation by relaxation of the bowels to a moderate degree, such as can be obtained by calomel (gr. ii. to v.) or croton oil (m i. to ii.), followed if necessary by an enema.

The patient's head, upon which an ice-bag may be placed, should be raised and turned rather to one side, in which position the stertor is often diminished. For the first few hours no food is necessary; after that a little milk may be given. When necessary the urine should be drawn off, and great care taken to keep the skin clean and dry.

Prognosis.—The next step which it is usually necessary to take is to give an opinion as to the danger to life, and in this connection the chief points to observe are—

- (1) The depth of the coma.
- (2) The course of the temperature.
- (3) Whether conjugate deviation of the head and eyes is present.

A deep *loss of consciousness* suggests that the hæmorrhage is either large or situated in some specially important part of the brain, as, for instance, the neighbourhood of the pons or medulla; in both cases life is in danger. Of particularly evil significance is a coma which deepens progressively, for it usually indicates that the bleeding is still going on, or that the blood is finding its way into the lateral ventricles. Generally speaking, if there are no distinct signs of returning consciousness in twenty-four hours the outlook is bad. On the other hand, slight loss of consciousness and signs that the coma is beginning to pass off are both satisfactory indications.

Immediately after the hæmorrhage has taken place there is a sudden fall in the temperature to two or three degrees below normal, which is followed by a rise above normal when the preliminary shock has passed off. If this rise keeps within the limits of one to two degrees above normal the outlook is favourable; but if the temperature is raised above this for any length of time, and shows signs of being raised still more, it is a sign that the patient will probably die.

Conjugate deviation of the eyes and head, especially if it persists, is a sign of bad omen.

Other symptoms, such as the state of the pupils and the presence of Cheyne-Stokes breathing, may also help in making a prognosis.

SIGNS OF HEMIPLEGIA

With return of consciousness the local effects of the lesion become more apparent, and when the hæmorrhage is in the region of the internal

capsule there are often sensory as well as motor symptoms.

1. **Motor symptoms.** — There will most likely be loss of power of the upper neuron type for voluntary movements in the face, arm, and leg on the opposite side to the lesion.

Face.—In examining the face there are three sets of movements to be considered, viz.:

(1) Unilateral voluntary movements, i.e.,
voluntary movements that can habitually be made on one side of the face, *e.g.*, drawing up the corner of the mouth.

(2) Bilateral voluntary movements, i.e.,
voluntary movements in which both sides of the face are habitually put into action at the same time, *e.g.*, closing the eyes and wrinkling the brow.

(3) Emotional movements, e.g., those of
laughing and crying, into which
volition only enters to a slight
degree.

In hemiplegia these three sets of movements are affected in very different degrees.

The unilateral voluntary movements are the most highly specialised, and the path by which impulses for their production can pass from the motor cortex to the muscles is confined to the direct course of the motor fibres. Interruption of these fibres leaves no alternative route, hence these particular movements are lost.

Bilateral voluntary movements are less specialised, and there is still some communication possible between the nuclei of their nerves on the two sides. When, therefore, the path from the cortex is interrupted on one side, it is still possible to obtain impulses through the communication with the nucleus on the other side, and so in hemiplegia the voluntary bilateral movements,

though often temporarily weakened, do not suffer to such an extent as the unilateral ones. The patient is thus enabled to wrinkle his brow, frown, and close the eyes, even though he cannot move the corner of his mouth.

Reference has already been made to the lateral deviation of the head and eyes which may take place in severe cases.

Emotional movements are still less diminished, and the corner of the mouth often moves freely during laughing or crying when it cannot be moved by the will.

The tongue, when put out, usually deviates towards the paralysed side, and on phonation the two halves of the soft palate are unequally drawn up.

The arm is generally deeply paralysed, the finer movements of the fingers suffering more than the coarser ones of the elbow and shoulder.

The leg is usually less deeply paralysed than the arm, and is nearly always the first to show signs of returning power.

The movements of the trunk are for the most part bilateral, and, although slight differences can be detected between the respiratory movements of the two sides, they are not usually of special clinical importance.

2. **The reflexes.**—The tendon reflexes on the paralysed side, as is the rule in upper neuron lesions, are exaggerated, and after a time there is sometimes an increase in those of the opposite side also. In the leg the knee-jerk is very active and ankle clonus can usually be obtained. The plantar reflex generally gives a typical extensor response.

After return of consciousness, unless there is considerable impairment of the mental faculties, the organic reflexes, such as those of the bladder and deglutition, are usually under control.

3. **Sensory changes.**—Hemianæsthesia may

be present when the sensory part of the internal capsule is injured, and, as the fibres for vision from the occipital lobes also pass over the same part of the internal capsule, hemianopia is often an accompanying symptom. In the majority of cases sensation is eventually restored, and the hemianopia also passes off.

4. **Mental changes.**—There is generally some degree of enfeeblement of the mind after a "stroke," and it is usually safe to predict that the patient will not retain the same acuteness of intellect that he possessed before the occurrence of the hæmorrhage.

Course of events.—As time goes on, the signs which accompany degeneration of upper neuron fibres become more apparent. The tendon reflexes become further increased, and ankle clonus and the extensor response of the plantar reflex become apparent if they do not already exist.

There is a tendency for the limbs to become rigid and to develop contractures. At the same time there is no obvious wasting, except such as may be accounted for by disuse, nor can any changes be observed in the electrical reactions of the muscles.

The onset of rigidity and contractures causes hemiplegic patients to assume a characteristic attitude and gait.

The contractors of flexors and adductors are stronger than those of extensors and abductors, so that the arm is drawn in close to the side, pronated, and flexed at the elbow, wrist, and fingers. The leg is generally much less contracted than the arm, but the rigidity prevents the joints being freely used, and in walking the whole limb is stiffly swung round in the arc of a circle from the hip, i.e., circumducted (Plate XVIII.).

The hypotheses which have been brought forward to account for this rigidity and the methods

necessary to adopt in order to avert it are discussed in Chapter III. (pp. 30-31).

Involuntary movements.—The hemiplegic condition is sometimes accompanied by involuntary movements known as post-hemiplegic chorea and athetosis. It is thought that these movements show a tendency to occur more particularly in lesions of the thalamus and its neighbourhood, but in many instances the disease is situated in the cortex. These movements are very apt to arise in the hemiplegias and diplegias of childhood, but their onset may be postponed for a considerable time after the date of the original lesion.

Post-hemiplegic chorea is characterised by tremors or irregular movements in the arm or leg of the paralysed side.

Athetosis is characterised by involuntary movements which are generally confined to the hand or foot, but occasionally the whole limb may be involved, as also may the face.

In the hand the fingers move in a slow manner, and are alternately flexed and hyper-extended. All the fingers do not move equally, but one or more at a time are hyper-extended in a fashion which it is not possible to imitate. In some instances the movements go on almost continually, but in others they are often started by an attempt to move them voluntarily. In severe cases the whole limb may be thrown about in such a fashion that it becomes necessary to bind it to the patient's side.

Pain.—In some cases pain in the paralysed limbs is a very distressing symptom. It is probably due to persistent irritation of the sensory fibres.

Associated movements are also met with in hemiplegics. The paralysed arm is often strongly drawn across the chest when the patient yawns or breathes deeply, and the movement of



PLATE XVIII —Characteristic Gait in Hemiplegia.
The rigid and weakened left leg is stillly circumducted from the hip.
Note also the characteristic position of the paralysed arm.

one leg may be accompanied by some movement of the other.*

When all signs of improvement in the power of the limbs cease, and rigidity makes its appearance, there is not much hope of any further improvement so far as recovery of the functions of the injured neurons is concerned. Treatment must, however, on no account be abandoned, for by improving the function of fibres that remain, and by warding off rigidity and contractures, much can be done to render the limb a useful one. If the methods described on pp. 30-31 are persevered with, striking results can often be obtained, and it must always be remembered that the difference between a helpless limb and one that can be used however slightly is of the greatest importance to the patient.

HÆMORRHAGE INTO THE PONS AND CRUS CEREBRI

Hæmorrhage into the pons is usually fatal within a few hours.

Deep coma, contracted pupils, and hyperpyrexia are among the characteristic signs. Convulsions may also occur. There is often paralysis on both sides of the body, since a hæmorrhage of any size will injure both sets of motor fibres.

If, however, the hæmorrhage is small and situated at the upper part of the pons or in the crus cerebri, it is possible to get "crossed paralysis," i.e., of the face on one side and of the arm and leg on the other, owing to the fibres of this nerve crossing at a higher level than those for the arm and leg, which do not decussate till they reach the medulla.

The fibres to the ocular muscles may also be affected in lesions of the pons.

The association of coma and contracted pupils makes it necessary to bear in mind the possibility

* A paper by the Author on the possible explanation of the origin of these associated movements will be found in *Brain*, Part civ., 1904.

of opium poisoning when no history of the case is available.

Summary of Causes of Hemiplegia.—

The symptoms of hemiplegia following cerebral hæmorrhage as described in the previous paragraphs will serve as a general example, but it is also necessary to remember the chief causes of this condition, since the patient may be seen for the first time long after the occurrence of the causal lesion.

The causes of hemiplegia may be classified somewhat as follows:

- | | | |
|--|---|---|
| 1. <u>Vascular lesions</u> | { | Hæmorrhage. { <u>Meningeal</u> .
<u>Intracerebral</u> . |
| | { | Thrombosis.
Embolism. |
| 2. <u>Inflammations</u> ... | { | Meningitis. { Localised, or especially
intense over the
motor areas. |
| | { | Acute encephalitis. |
| 3. <u>Tumours</u> ... | | Pressing on the motor tracts. |
| 4. <u>Degenerations</u> ... | | Degenerative processes of the brain,
e.g., <u>sclerosis</u> . |
| 5. <u>Toxæmias</u> | | In <u>uræmia</u> , the effects of the poison
are sometimes especially visited on the
motor areas, and a hemiplegia which
is often difficult to distinguish from
that of cerebral hæmorrhage may
occur. |
| 6. <u>Exhaustion</u> fol-
lowing <u>convul-</u>
<u>sions</u> . | { | Temporary hemiplegia, presumably due
to exhaustion, may follow a severe
epileptic fit. |
| 7. <u>Functional hemi-</u>
<u>plegia</u> . | { | Hemiplegia is a not uncommon mani-
festation of hysteria. |

CEREBRAL THROMBOSIS

Etiology.—The chief conditions which lead to thrombosis are atheroma of the vessels and a feeble circulation; hence, thrombosis is more likely to occur in advanced years, when the vessel walls are thickened and the circulation slow. On the other hand, in the latter part of middle

life, when the arteries are apt to be weakened, while the force of the heart still remains strong, there is a greater liability to hæmorrhage.

Among the principal causes of arterial degeneration which may lead to thrombosis are syphilis, alcohol, worry, and Bright's disease, and of these syphilis is the most important. The atheroma of Bright's disease is more likely to be associated with hæmorrhage than with thrombosis.

Syphilitic endarteritis is the commonest cause of the cases of cerebral thrombosis that are met with prior to the occurrence of the degenerative changes of senility. The proliferation of the tissues of the arterial coats gradually narrows the lumen of the vessel until finally the flow of blood is checked and the parts beyond are deprived of their nutrition.

In some instances the cause of the thrombosis depends upon changes in the composition of the blood. This is the case in thrombosis associated with severe anæmias and with some varieties of septicæmia. Allusion has already been made to the infective processes as a cause of thrombosis in infantile paralysis and the cerebral palsies of children.

Symptoms.—The symptoms vary according to the mode of onset and the locality of the vessel that is blocked.

1. Gradual onset.—When the lumen of the vessels becomes narrowed gradually, the first symptoms are likely to be those due to diminished blood supply to certain parts of the brain, *i.e.*, to cerebral anæmia, and the function of the part is then apt to be impaired without being altogether lost, and it may again perhaps be restored as some variation takes place in the state of the local circulation. Thus, before there are signs of complete thrombosis, there may be warnings in the guise of temporary failure of power in a limb, an ocular muscle, or an eyelid, or there may perhaps

be some loss of memory, with a passing condition of partial aphasia; while in other cases the patients complain of tingling, numbness, and giddiness. One day matters may go further: a clot forms, and the area supplied by the vessel is cut off from the circulation.

If, as frequently happens, a branch of the middle cerebral artery to the motor cortex is blocked, there will be loss of power corresponding to the extent of the brain affected. If, for example, the branch that is blocked happens to be that supplying the motor area of the cortex in which movements of the arm are represented, the limb will be paralysed, and will also very likely be subject to attacks of localised convulsions owing to the irritation of the cerebral cortex.

If a branch of the lenticulo-striate artery is thrombosed, the internal capsule will be affected, and there will be hemiplegia, with or without hemianæsthesia and hemianopia, according to the extent of the lesion.

When a clot has formed it frequently extends into other branches, and thus a monoplegia may in a few hours be succeeded by a hemiplegia.

With a gradual onset the general symptoms of cerebral disturbance are not likely to be great, and in many instances consciousness is never lost.

Where the tendency to thrombosis is widespread, loss of memory and general mental failure may be the most prominent features of the case.

2. *Sudden onset*.—A more sudden onset is the characteristic of other cases where the function of the part has not been perceptibly impaired before the vessel becomes completely blocked. Paralysis of one or more limbs sets in suddenly, and the general cerebral disturbance is then often sufficiently great to cause loss of consciousness, though the coma is seldom so deep as that of a hæmorrhage causing symptoms of a corresponding

extent. Some idea of the effects of thrombosis of different arteries and their branches can be gauged from the diagram which shows the area of softening after blocking of the middle cerebral (Fig. 84, p. 347).

The middle cerebral artery supplies nearly the whole of the motor area of the brain, cortical and subcortical, and a complete hemiplegia, together with aphasia when the lesion is on the left side, results when its main trunk is blocked. An equally severe hemiplegia may also result from thrombosis of the vessels supplying the motor fibres of the internal capsule, but speech is not in these circumstances often permanently lost.

Pathology.—An area of the brain that is deprived of its blood supply becomes softened and necrosed. Sometimes it remains pale, but often there is some transudation of blood, as in the case of infarcts of other organs; and pale, red, and yellow softening are spoken of, according to the coloration produced by the blood. In late stages a cyst or a scar may represent the area of disease.

Prognosis.—In giving a prognosis of the results of thrombosis, it is necessary to try to form a mental picture of what is taking place at the seat of the disease. The blockage of an artery causes total anemia of that portion of brain which is entirely dependent on it for its nutrition, and partial anemia of the parts around, which can still obtain some nutrition from collateral sources.

Hence the first effect is destruction of the function of the nerve cells in the area which the vessel wholly supplies and a lowering of the function of those parts on the border of that area which obtain some of their blood supply from elsewhere. Unless the circulation to the part which is wholly dependent on the thrombosed artery is restored quickly, the nervous tissue will disintegrate and be incapable of repair. On the other hand, the function of the parts in

the immediate vicinity, depressed as the first effect of the arterial blockage, may improve to a considerable extent as the collateral circulation becomes better established. So that when, after a few days, there is no return of power, it may be fairly assumed that permanent damage has been done, though a certain degree of recovery may still take place as the function of the surrounding part improves.

Thrombosis of the vessels supplying the region of the internal capsule is especially apt to be followed by permanent softening, for the collateral circulation of that part of the brain is very poor and can only be very feebly maintained from the terminal branches of the cortical arteries.

The outlook is bad in those cases in which gradual failure of the mind signifies a widespread softening.

Diagnosis.—The diagnosis from cerebral hæmorrhage has already been considered (p. 316).

Treatment.—If the danger is recognised in time, as it may be when the circulation is weak and the arteries obviously thickened, much can be done to avert thrombosis by increasing the rate of the blood-flow and by modifying the nutrition of the vessel walls; but, once the clot has formed, no method of treatment will ensure its dispersal, nor can any drug restore the vitality of a patch of softened brain.

In those who have syphilitic endarteritis, a course of mercury and iodides would doubtless often prevent catastrophe, but too often they seek no treatment till the event has taken place, and the most that can usually then be done is to try to prevent a recurrence.

In every recent case of thrombosis due to syphilitic disease of the arteries it is a good plan to prescribe at once a course of mercury and iodide, and afterwards, with a view of anticipating recurrence, to give regular courses of iodide at

stated intervals, to which, if it seems desirable at any time, a course of mercury may be added.

During the attack the patient should be kept quiet and fed lightly. Care should be taken not to purge severely and not to adopt any other measures that are likely to depress the force of the circulation, which may often be improved with advantage by a little digitalis or other suitable cardiac stimulant.

CEREBRAL EMBOLISM

Etiology.—Embolism is nearly always associated with endocarditis of the aortic or mitral valve. Occasionally it arises from detached portions of clot from an aneurysmal sac.

In chronic endocarditis, mitral stenosis is the form most likely to cause embolism, but in the acute infective forms a fragment may be dislodged from either the aortic or mitral cusps.

Emboli are on the whole more likely to pass up the left carotid artery, and so it is more common to get a lesion of the left than the right side of the brain.

Symptoms.—The onset is quite sudden and accompanied by symptoms which vary according to the position in which the clot lodges. If, as often happens, the main branch of the middle cerebral artery on the left side is blocked, hemiplegia and aphasia immediately result. In such a case the senses are often lost, but there is seldom the depth of coma, stertorous breathing, or disturbance of temperature that is met with in cerebral hæmorrhage.

Softening of the cortex will probably be accompanied by convulsions which will be absent when the obstruction occurs in the central branches of the artery after they have left the main stem, though the hemiplegia may be complete from softening of the fibres of the internal capsule.

When only a small branch of the middle

cerebral is blocked the symptoms are, of course, less severe. Thrombosis of the posterior cerebral artery causes hemianopia, and (according to Marie) visual aphasia also.

If thrombosis starts from the original clot and extends along the vessels, the sudden symptoms at the beginning are followed by others of more deliberate progress.

Prognosis.—What has been said regarding the prognosis in cases of cerebral thrombosis may be in a large measure applied to cerebral embolism. In both the restoration of cerebral function depends upon the possibility of the rapid re-establishment of the circulation, for when softening has once occurred in a part, its function cannot again be regained, though it may perhaps be partially compensated by the action of other parts.

Diagnosis.—The diagnosis between hæmorrhage, thrombosis, and embolism is discussed on p. 316.

Treatment.—The clot cannot be modified in any way. The after-treatment is that detailed for hemiplegia, and consists in massage and correction of faulty positions of the paralysed limbs, while the patients must be encouraged to further any return of voluntary power.

CHAPTER XXXIII

THROMBOSIS OF CEREBRAL SINUSES

THROMBOSIS of the cerebral sinuses may arise as the result of general diseases, e.g., severe anæmia, wasting and diarrhœa (especially in infants), and typhoid fever, or it may take place after a local injury. In these cases the thrombosis is often called "simple," as opposed to the infective variety, which is the more important, and which arises as the result of some near or distant septic condition. Suppuration of the middle ear and nasal sinuses is the most important local cause of infection.

Thrombosis of the lateral sinus is especially apt to be associated with chronic suppuration of the middle ear, and it may coexist with cerebral abscess.

The chief local signs are pain, œdema, and distension of veins over the mastoid region, with a sense of hardness over the course of the internal jugular vein, down which the clot frequently extends. The general symptoms are mainly those of septic infection, viz., high temperature, rapid pulse, and delirium.

This condition gives cause for grave anxiety. The best mode of treatment is to open up the sinus and clear out the infective clot, and at the same time to deal with an abscess, should it be present.

Thrombosis of the cavernous sinus.

—St. Clair Thomson* has shown that thrombosis

* *Trans. of Med. Soc. of London*, 1906.

of the cavernous sinuses is specially liable to follow chronic suppuration of the nasal cavities.

The main *general symptoms* which indicate the onset are, according to Thomson, acute head-



Fig. 77.—Edema and exophthalmos in a case of thrombosis of the cavernous sinus. (St. Clair Thomson.)

ache, delirium, and drowsiness, with sickness, high temperature, and rapid pulse. The headache is generally severe, and the pain may be referred to the side of the head, to the back of the ear, or to the frontal region. With these there are generally

some ocular symptoms, consisting chiefly of papillary oedema, chemosis, and exophthalmos, to which may be added ulceration of the cornea and paralysis of ocular muscles from implication of the first division of the trigeminal nerve and of the motor nerves in their passage along the outer wall of the sinus (Fig. 77).

Swelling of the optic disc is also often found, but, if the case runs its course rapidly, many of the ocular symptoms do not develop.

The prognosis in a case of thrombosis of the cavernous sinus is very grave, and still more so if the condition of the eyes indicates bilateral disease. Suggestions and attempts have been made to reach these sinuses, but they have hitherto met with little success.

Thrombosis of the longitudinal sinus generally complicates some general debilitating disease and seldom arises from direct infection. It may be met with in weakened and wasted children, after typhoid fever, or it may complicate severe forms of anæmia.

The symptoms mostly depend upon interference with the cerebral cortex, and among them are mental dulness, convulsions, and possibly paralysis if the vascularity of the motor areas is modified. In many cases the symptoms are obscured by those of the previously existing general condition.

CHAPTER XXXIV

GENERAL PARALYSIS OF THE INSANE

GENERAL paralysis of the insane is seen by the neurologist chiefly during its early stages, when it is most important, if possible, to arrive at an accurate diagnosis.

Etiology.—Men are more liable to suffer from the disease than women, and it most commonly begins between the ages of 35 and 55. Previous syphilis can be traced in a large number of cases, and it has now become customary to regard the pathological process as very similar to that which occurs in locomotor ataxy, *i.e.*, to suppose that the syphilitic poison modifies the nutrition of the nerve fibres in such a way as to make them prone to degenerate on the least provocation. In this way injury, alcohol, and mental worries may upset the balance of nutrition. Symptoms of locomotor ataxy and of general paralysis are often found together in the same patient.

While it seems fairly certain that syphilis often conduces to the breakdown, there is good reason to believe that it is not the only poison concerned, for there still remains a small percentage of cases in which no history of syphilis can be traced, even after ample allowance has been made for the possibility of infections of slight intensity which have passed unnoticed.

Recent observations, as mentioned under the heading of Locomotor Ataxy (p. 169), have been made with a view of showing that general paralysis

is due to an acute infection, and that the organism is a bacillus which closely resembles that of diphtheria.

Pathology.—To the naked eye, atrophy of the cerebral convolutions, especially those of the frontal lobes, together with thickened and adherent membranes, are among the principal changes. With the microscope, degeneration of cells and tracts of fibres connecting various parts of the cortex with one another can be demonstrated, and chemical observations have shown that the products of degeneration act as poisons to the still healthy fibres, and thus a circle of events is established which ultimately produces complete physical and mental wreck.

Symptoms.—The first symptoms are usually those connected with the mind. The patient becomes irritable, unstable, and alternately unduly depressed and exalted. Early mental changes vary in character in different individuals: in some patients depression is a feature; in others exaltation is present. Convulsive seizures, though commoner in the later stages, are sometimes among the first symptoms to be noticed.

Forgetfulness is especially apt to be noticed as an early symptom when the patient is a business man, for naturally any mistakes are soon liable to be found out; but where the patient has not a responsible position, loss of memory, although perhaps present, is more likely to be overlooked. The same is true of manual work; the last and most delicate acquirements are the first to fail, on account of the failure of the attention and the delicate co-ordination that are required for their performance.

When the patient is brought for examination there are frequently definite signs which, taken with the mental condition, make the nature of the case clear. Of these the most common are inequality of the pupils with failure to react to

atrophy of cerebral convolutions spec. frontal lobes. thickened & adherent membranes.

Degeneration of cells & tracts Connecting various parts of the brain & health of the brain.

light, a "shurring" speech, and fine tremors of the lips, tongue, and hands.

The pupils are like those of locomotor ataxy (Argyll-Robertson), and contract during accommodation but not to light. Their inequality is often marked, and they have often ceased to be circular, and exhibit an "angular" appearance. There is no nystagmus, and in the early stages the optic discs do not usually show any change, although later on they may undergo atrophy.

The speech is generally tested by the ability to say such words as "artillery," "constitutional," and "constabulary." There is a great tendency to elide syllables, a tendency which is also noticed

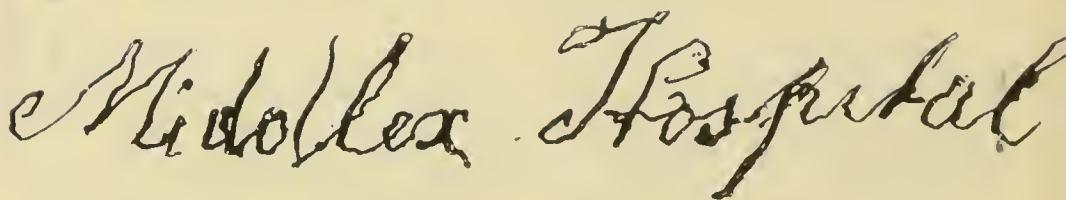


Fig. 78.—Specimen of writing from a case of general paralysis.

in the writing. When talking there is a fine tremor of the lips and a general over-action of all the face muscles; a fine tremor may also be observed in the tongue.

The hands tremble, and there is loss of accuracy in movements requiring fine co-ordination, as can often be demonstrated by asking the patient to button up his coat.

The tremor, incoordination, and general failure of intellect are often all made manifest in the writing.

The character of the knee-jerks varies; they may be absent or increased, and in the latter event they are often unequal.

Diagnosis.—In the early stages, care must be taken to avoid confusion with neurasthenia. Occasionally it may at first be impossible to make a certain diagnosis, but usually there is a

difference between the simple fatigue symptoms of neurasthenia and the mental degeneration of general paralysis; and with the development of physical signs the diagnosis becomes clear. The neurasthenic worries about himself, but in general paralysis it is the friends who are often first worried on account of the mental symptoms which change the man from his former self into an irritable, careless, and otherwise strange being.

This disease may also bear some resemblance in its early stages to disseminated sclerosis, the chief symptoms common to both being tremors, alteration in speech, and increase of deep reflexes; but it must be remembered that the knee-jerks are by no means always increased in general paralysis—they may be diminished, unequal, or absent. The tremor of general paralysis is much finer than that of disseminated sclerosis, and it is, moreover, especially marked in the tongue and lips. The speech, too, when well developed, differs markedly in the two conditions; in general paralysis it is hesitating, and syllables are dropped or telescoped into one another, while in disseminated sclerosis it is jerky, staccato, or scanning—characters which are rather difficult to describe, but which are distinctive when heard. Among other conditions which may simulate general paralysis are some phases of mental disorder due to *chronic alcoholism*, and some cases of *syphilitic* (probably gummatous) *affections of the brain*. Tumours of the frontal lobes with mental symptoms and tremors may also bear some resemblance, but the presence of optic neuritis and the absence of Argyll-Robertson pupils serve as distinguishing features.

Prognosis.—The prognosis is always bad, and the patients, with the exception of occasional curious remissions, tend to go steadily downhill. Fits, epileptiform or apoplectiform in character, are often a marked feature, and each attack generally leaves the patient both physically and

mentally worse than he was before. As the mind deteriorates the physical weakness increases, until at last the patient arrives at the state which justifies the use of the term "general paralysis."

Treatment. — In the early stages anti-syphilitic remedies may be tried, but at present no method by which the disease can be arrested is known, and the treatment resolves itself into placing the patient under adequate care and control and attending to the general health.

The conclusions which have been arrived at by Ford Robertson and Douglas McRae concerning the infective nature of general paralysis have led to the trial of treatment by serum. It is not possible to speak positively at present on the general value of this method, but results which show promise have already been obtained, and it is not too much to hope that these researches will eventually lead to a more complete understanding of the cause and treatment of this disease.

CHAPTER XXXV

APHASIA

THE generally accepted views concerning the acquisition of powers of communication by language can most easily be understood by considering the education of a child. The first step is the recognition of certain sounds which, by repetition, come to be associated with certain objects. Thus the memory for words is first developed in an area of brain cortex situated in the first temporal convolution of the left side.

After a time the child ceases to be content to hear words, but wishes to produce them also, and after repeated trials succeeds in doing so by developing an existing (inherited) track to a portion of the cortex connected with movements of the tongue and lips. This part is situated in the posterior part of the third frontal convolution of the left side, and is known as Broca's convolution.

The child is now able to understand what is said and to make himself understood by others (Fig. 79).

Further development takes place by teaching the child to read and write.

First the letters have to be recognised by educating the visual centre in the angular gyrus, and afterwards they have to be reproduced by the education of the writing centre, which is situated (probably) in the ascending frontal convolution close to the centre for other movements of the hand (Fig. 80).

word memory
1st Temporal
Left side

Speech
Post. part of
3rd Frontal
Left side

Visual Centre
angular gyrus

Writing Centre
Ascending Frontal

We have now the two pairs of centres educated thus. And to make the powers of communication still wider, further education makes it possible for impulses to pass from the auditory to the visual centres and *vice versâ*, thus making it possible to read aloud (in which the impulses pass from the visual to auditory, to Broca's convolution) and to write from dictation (in which impulses pass from auditory to visual, to writing centres) (Fig. 81).

In right-handed people those centres are, as a

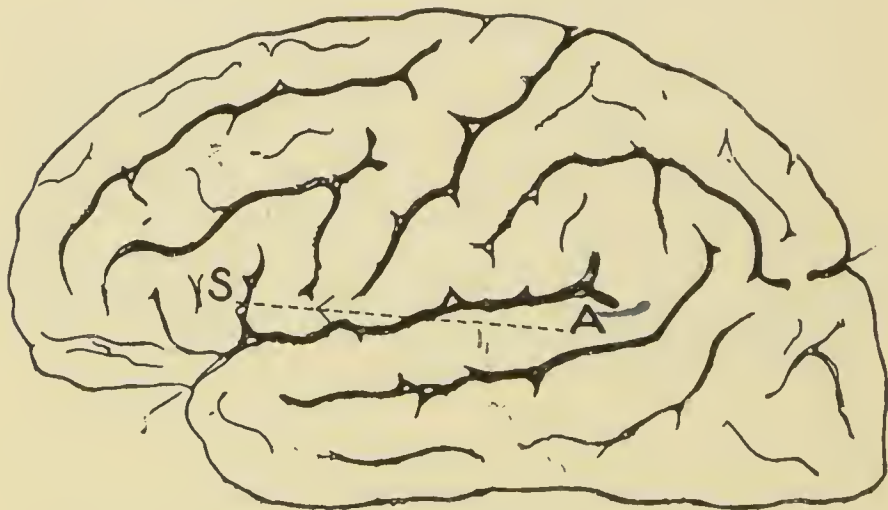


Fig. 79.

rule, all situated in the cortex of the left hemisphere, but in those who are left-handed the position is generally reversed.

In examining a case of aphasia, the function of these four centres and the integrity of their commissural fibres should be tested in turn, and the following method is based on the plan adopted by C. E. Beevor :

A. Test the first pair of centres, viz., the motor and auditory centres, and their commissural fibres (Fig. 79).

1. The motor centre (S).—Can the patient speak intelligently? This tests the condition of the motor mechanism at Broca's convolution. If

this centre is destroyed he loses the power of co-ordinate speech, and is unable to repeat the names of words or objects though he may still be able to say a few words, usually the same ones over and over again. This condition is known as **motor aphasia**.

2. **The auditory receptive centre (A).**—Can the patient understand what he hears? Speak some simple command, such as "Stand up," or "Hold up your hand." This tests the condition

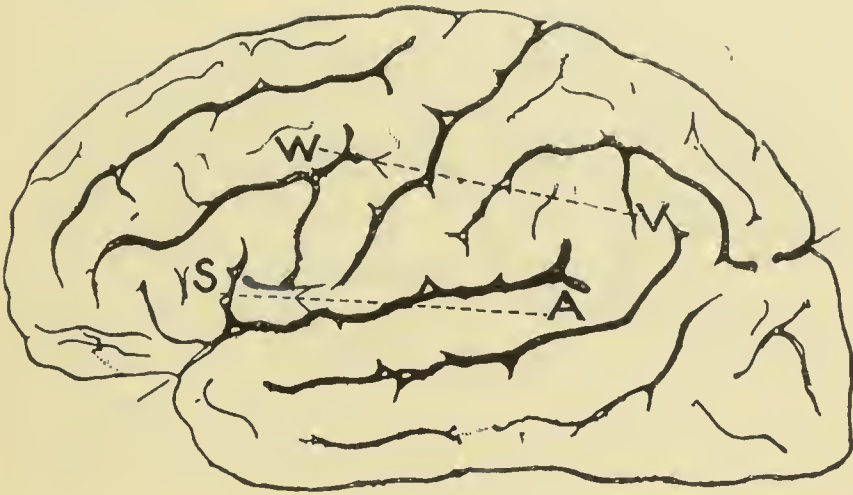


Fig. 80.

of the auditory receptive centre. If this centre is destroyed, sounds lose their meaning and convey no understanding—a condition spoken of as **word-deafness**.

3. **Communication between auditory and motor centres (A-S).**—Can he repeat names of things or letters that he hears? This tests the condition of the fibres between the auditory and the motor centres.

B. Next test the **second pair**, viz., the visual and writing centres and their commissural fibres (Fig. 80).

1. **The visual centre (V).**—Can the patient understand written language? Place be-

fore him some written request, such as "Stand up," or "Hold up your hand." This tests the visual centre in the angular gyrus, and, if its function is impaired, the letters, though seen, convey no intelligible meaning, and the condition is then spoken of as **word-blindness**.

2. **The writing centre (W).**—Can the patient write spontaneously? This tests the centre for writing, which is probably situated near or in the area for that of movement of the hand.

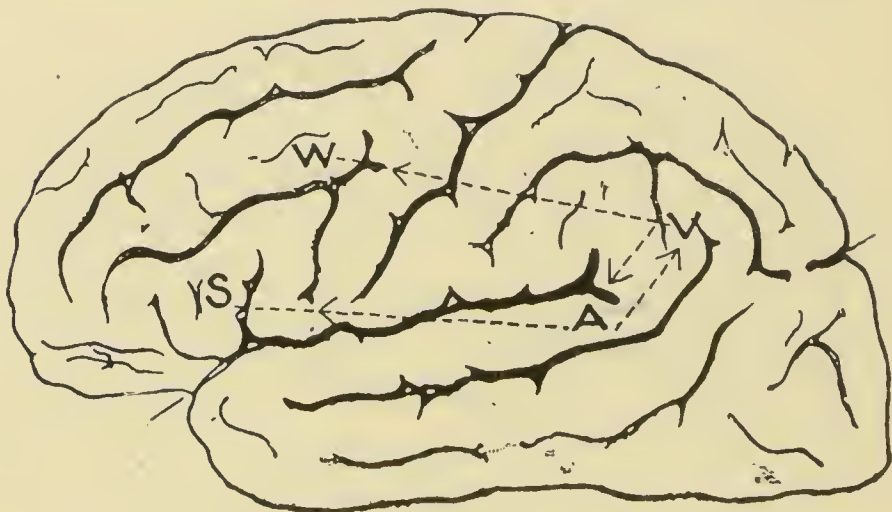


Fig. 81.

Failure is known as **agraphia**. This test is often difficult to apply, first, because the auditory or visual centre has to be intact in order to convey the command, and secondly, because paralysis of the right hand, which is so frequently associated with aphasia, makes writing impossible. It is only very rarely that agraphia exists alone.

3. **Communication between the visual and the writing centre (V-W).**—Test the commissural fibres from the visual to the writing centre by asking the patient to copy from printed to written characters, which entails a visual impression that has been understood passing to the centre for hand movements.

C. Test the communication between the **auditory and visual centres**—from the auditory to visual, to writing centres; from the visual to auditory, to motor speech centre (Fig. 81).

1. **Communication between auditory and visual centres.**—Test the fibres which connect the auditory and the visual centres (A-V), and for this purpose ask the patient to pick out letters or objects the names of which he hears. This entails the impulses passing from the auditory to the visual centres and being comprehended at both.

2. **Communication from auditory to visual, to writing centres (A-V-W).**—Test this by asking the patient to write from dictation.

3. **Communication from visual to auditory, to motor speech centre (V-A-S).**—Test this by asking the patient to read aloud and understand what he is reading.

If these tests for the different centres are applied methodically, noting after each where the patient succeeds and where he fails, a good idea of the extent of the lesion will generally be obtained, but in practice the investigation is often made difficult by the partial or complete destruction of more than one centre.

Individuals also vary considerably as regards the effect of partial lesions. Failure to read (*alexia*) especially seems to vary in this respect, and is no doubt often influenced by the degree to which the patient has been educated. Where a patient knows several languages, a comparison sometimes shows that he has retained more command over some than others.

Also, from subcortical lesions, the impulses may be prevented from reaching a centre though the centre itself remains intact. In the case of this happening to the auditory centre, the patient may be "word-deaf," though he can still talk, since his memory for words exists. Similarly, in the case

of the visual centre, the patient may be unable to recognise printed words and yet be able to express himself in writing.

It is, of course, important not to confuse a peripheral lesion which impairs the articulation of language with aphasia.

Etiology.—Aphasia is produced by lesions of the cortex, and the commonest cause of these is thrombosis of the middle cerebral artery or some of its branches. Embolism and cerebral tumours are also important causes. Hæmorrhage into the cortex is uncommon.

When a lesion occurs in the course of the motor fibres below the level of the cortex, there may be difficulty in speech due to the inability of the impulses to pass down from the cortex, and in some instances this may amount to a complete pure motor aphasia, the power of understanding remaining unimpaired.

Recent Views concerning Aphasia.*—

Pierre Marie has recently questioned the correctness of the currently accepted ideas on aphasia, and in view of the importance of his work and of the further researches which his contributions to the subject are certain to promote, it is necessary to give a brief account of the conclusions at which he has arrived.

Marie maintains that in every case of aphasia due to a cortical lesion there is some degree of intellectual impairment, as shown by the failure to comprehend spoken language, and that the omission to observe this fact hitherto has been due to the too simple character of the tests that have been applied.

Further, Marie denies the existence of the several independent centres described in the preceding section, and recognises one form of aphasia only, viz., that which is brought about by a

* An account of Professor Pierre Marie's views on this subject will be found in *La Semaine Médicale*, 1906.

lesion in the zone of Wernicke, comprising the supramarginal convolution, the angular gyrus, and the base of the first and second temporal convolutions (Fig. 82). This aphasia, which, as already stated, is invariably characterised by some degree of impairment of the understanding, may be further complicated by loss of power to speak, which loss is produced by a subcortical lesion, and is known as anarthria. The degrees in which the

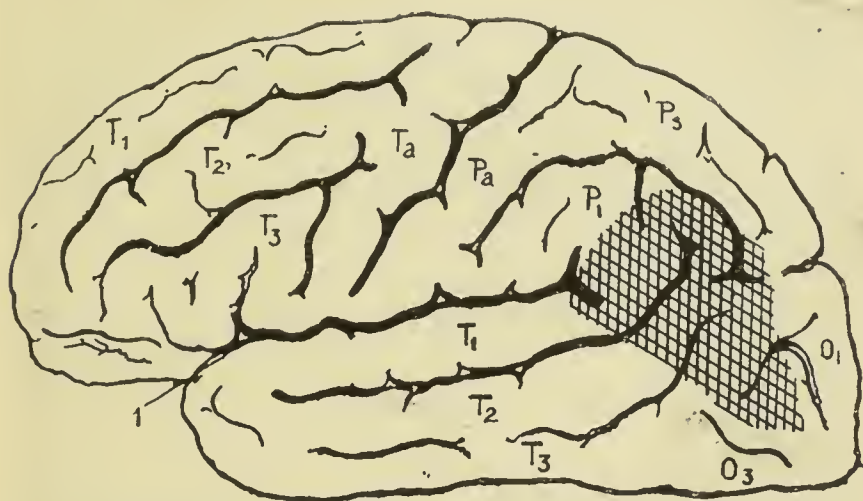


Fig. 82.—Lesion of angular gyrus and bases of two upper temporal lobes accompanied by aphasia of Wernicke. (*Pierre Marie.*)

powers of understanding and speech are impaired will, of course, differ with the extent of the lesion.

Marie brings forward strong evidence to show that Broca's convolution has nothing whatever to do with the function of speech, and cases are described in which tumours and other lesions of this area were totally unaccompanied by any motor aphasia (Fig. 83). It is not, of course, denied that aphasia is often associated with lesions of Broca's convolution, but a close examination of such instances shows that the destruction of tissue is not strictly limited to this area.

The three terms, Broca's aphasia, Wernicke's

aphasia, and anarthria, are described by Marie as follows:

(1) In the **aphasia of Wernicke** the patients can speak, but their intellect is always impaired to a variable extent, so that they may talk nonsense; there is also loss of the power of reading and writing, and an incapability of com-



Fig. 83.—Cortical lesion at the level of the third left frontal convolution in a right-handed man, without any disturbance of speech. (*Pierre Marie.*)

prehending spoken language.

(2) In the **aphasia of Broca** the patients can neither read nor write, neither can they comprehend fully all that is said to them. So far, they resemble the cases of aphasia of Wernicke, but they have the additional symptoms of being unable to speak.

(3) In **anarthria** there is simply an inability to speak from interruption of the motor fibres below the cortex. This disability is met with

in association with lesions of the lenticular zone; and Marie maintains that the aphasia of Broca consists of the aphasia of Wernicke together with anarthria, and that it is not in any way connected with lesions of the third left frontal convolution.

And thus it comes about, as stated above, that Marie recognises only one form of aphasia, viz., the aphasia of Wernicke, which he localises in the supramarginal convolution, the angular gyrus,

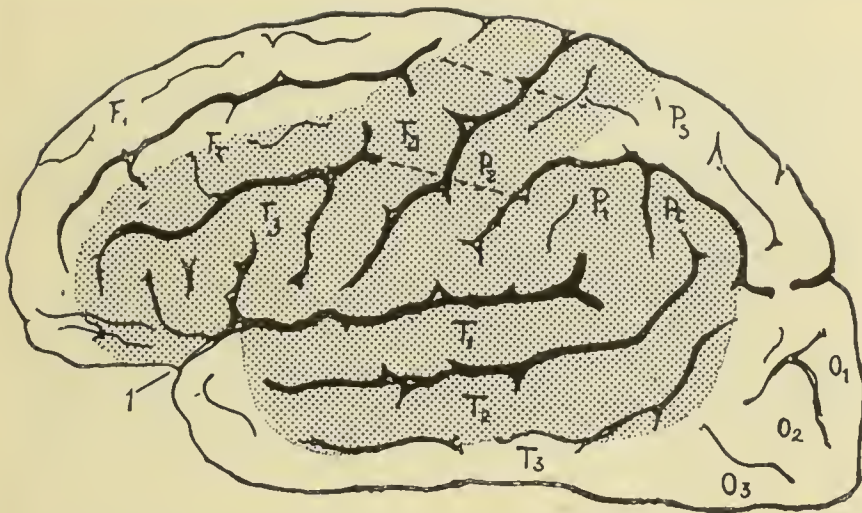


Fig. 84.—Softening of an extensive area of the cortex due to obliteration of the Sylvian artery at its commencement. The patient had complete aphasia of Broca, but the area of Wernicke and the central ganglia were softened also, and it is to the destruction of these that Marie attributes the aphasia in this case. (*Pierre Marie*.)

and the base of the first and second temporal lobes, known altogether as the zone of Wernicke, and he considers that destruction of this area gives rise to the aphasia of Wernicke (*i.e.*, the inability to understand spoken language). If, at the same time, the fibres proceeding from the cortex are also interrupted, then there is, in addition to the inability to understand spoken language, also the inability to speak known as anarthria, and the two together (*i.e.*, the aphasia of Wernicke and the anarthria) make up the aphasia of Broca.

The area of Wernicke is affected when the middle cerebral artery is blocked.

Word-blindness appears to depend upon a lesion of the posterior cerebral artery, and is associated, according to Marie, with destruction of the lingual and fusiform lobes on the inferior surface of the brain, though it may perhaps occasionally be possible for an extension of the softening due to a lesion of the middle cerebral artery to produce this symptom.

Marie sums up his views by dividing the varieties of aphasia into the following groups:

1. *Intrinsic aphasia*, in which the zone of Wernicke (*i.e.*, the zone of language) or the fibres which spring from it are directly affected by the lesion, and in which the patient is unable fully to understand spoken words.

2. *Extrinsic aphasia*, where the lesion may be in the lingual and fusiform lobes and give rise to word-blindness (*alexia*), or in the region of the lenticular nucleus, giving rise to *anarthria*, which is the inability to speak though everything heard can be clearly understood. This last type includes those cases of pure motor aphasia the lesion for which has hitherto been placed in the third left frontal convolution. As already stated, if the *anarthria* is also accompanied by a lesion of Wernicke's zone, the result is an inability to speak and also a failure to understand.

SECTION VI.—DISEASES OF FUNCTIONAL AND GENERAL ORIGIN

CHAPTER XXXVI

MYASTHENIA GRAVIS (ASTHENIC BULBAR PARALYSIS)

THE characteristic symptom of myasthenia gravis is the ease with which muscles become exhausted. Partial recovery of power often occurs after a period of rest, but the affected muscles are, as a rule, incapable of carrying out a sustained movement for any length of time.

Etiology and pathology.—The sexes suffer in about equal proportions, and in most cases the symptoms occur during the first half of life. There is still a considerable degree of doubt concerning the morbid conditions which underlie the symptoms.

So far as the central nervous system is concerned, beyond the occasional observance of some indefinite and inconstant changes in the nuclei of the cranial nerves, nothing notable has been recorded.

General opinion at the present time favours the view that the essential changes occur in the muscles themselves, and in support of this hypothesis is the finding by Weigert of an exudation of lymphoid cells in the muscle tissue, an observation which has been confirmed by Hun, Goldflam, Farquhar Buzzard, and others. It was present also in one of the cases which the author recently had an opportunity of examining.

Another abnormality frequently found is an enlarged thymus gland which is sometimes of a

lympho-sarcomatous character, but the connection, if any, existing between this and the exudation of lymphoid cells into the muscles is uncertain. At present one must be content to note the lesions and to assume that the disease depends upon some toxin, the origin and composition of which are as yet unknown.

Symptoms.—The symptoms are brought about by failure of muscular power, but, as this



Fig. 85.—Enlarged thymus gland in a case of myasthenia gravis.

is unequally distributed, they vary in different cases according to the parts affected.

There are also great fluctuations in the intensity of the symptoms, alternate exacerbations and remissions being very characteristic of the disease. The “ocular” and “bulbar” distributions are common.

In the case of the ocular symptoms, ptosis is generally a prominent feature, and the patients complain that the eyes cannot be kept open for long at a time. The lids persistently droop, and attempts are frequently made to correct the

inconvenience by throwing back the head or even holding up the eyelids with the fingers. A night's rest often partially restores the power, which again wanes as the day wears on, and any sustained movement, such as looking upwards for any length of time, rapidly exhausts the muscles and brings on the drooping.

The compensatory action of the occipitofrontalis, seen in the wrinkling of the brow, which is a usual feature of ptosis from other causes, is generally absent, since the muscle is quickly exhausted by any attempts at continuous contraction.

Transient ocular palsies of varying nature are common, and occasionally nystagmus is present.

The bulbar symptoms appear chiefly in connection with the orbicularis oris and the muscles of the tongue and palate.

Weakness of the orbicularis makes it difficult to blow or whistle, and also affects the speech. |||

The tongue when exhausted cannot be properly thrust out or forcibly protruded into the cheek. The paralysed soft palate ceases to shut off the nasal from the oral cavity, and so the nasal voice is produced, and at the same time there may be difficulty in swallowing with regurgitation of fluids through the nose. All these muscles become weaker after use, and exhaustion of the masseters, which often takes place during mastication, may make it impossible to finish a meal. The facial muscles may also suffer, and the weakness of the muscles round the mouth often causes an alteration in the expression which closely simulates that associated with the myopathies.

Another characteristic is the "nasal smile," in which the movement at the corners of the mouth is deficient, the furrow of the smile being sometimes entirely above the upper lip (Gowers).

When the limbs are at fault, walking causes

fatigue in a very short time, and no occupation involving the use of the arms can be engaged in for any length of time. In the trunk, the muscles of the neck often suffer and the head falls forward on the chest. Weakness of the muscles of respiration causes dangerous attacks of dyspnoea.



Fig. 86.—Bilateral ptosis in myasthenia gravis.

Electrical reactions.—The rapidity with which the power of the muscles is exhausted by repeated contractions can be demonstrated by stimulating them with the faradic current. After a variable number of contractions have been induced the response becomes gradually weaker and weaker, and finally ceases altogether, but may again be renewed after a period of rest. After the muscle has ceased to respond to the electrical stimulus it

may still be capable of voluntary contractions. This condition is known as the myasthenic reaction, and when present is very characteristic of the disease; but, like the reaction of the muscles from voluntary movements, it varies from time to time, nor is it necessarily equally obvious in all the affected muscles at one time.



Fig. 87.—Ptosis and general facial expression in myasthenia gravis. Note the absence of any compensatory act on of the occipito-frontalis. (From a case under the care of Dr. George Johnston.)

Reflexes.—The reflexes differ in their reactions according to the condition of the muscles which take part in their production. The knee-jerks are sometimes absent, or they can be exhausted by repeatedly eliciting them. Wasting of muscles is uncommon, but has occasionally been observed.

Sensory changes.—While the main symptoms are motor, there are occasionally some sensory changes to be noted. Aches and pains are liable

to occur, and Farquhar Buzzard* has recorded cases in which the pains closely resembled in their intensity those of locomotor ataxy. The same observer has also shown that analgesia and anæsthesia may be found, and that the presence of mental symptoms is not unknown.

Diagnosis.—For purposes of diagnosis the cases of myasthenia gravis may be divided into three groups, viz., ocular, bulbar, and spinal, according to the parts in which the early symptoms predominate.

1. Ocular type.—When ocular symptoms are the prominent feature, other conditions, in which ptosis and partial ophthalmoplegia externa are likely to occur, must be considered. In tabes when these symptoms are present there are almost certainly other distinctive signs, such as an Argyll-Robertson pupil, in association with lightning pains, loss of knee-jerks, and incoordination.

Transient ocular symptoms are sometimes associated with migraine, but here they generally bear some definite relation to the headache.

Cerebral tumours, syphilitic disease, and hysteria must also be considered as causes of ptosis. In hysteria the ptosis is usually spasmodic rather than paralytic, but outwardly it may show some similarity to that of myasthenia by the absence of the compensatory furrows.

2. Bulbar type.—When signs of bulbar trouble are prominent, progressive bulbar paralysis and post-diphtheritic neuritis will suggest themselves; in both these conditions the distribution of symptoms is apt to be similar. In progressive bulbar paralysis the early and pronounced wasting, with no remissions and absence of the myasthenic reaction, will serve to enable the distinction to be made.

In post-diphtheritic neuritis the history and course are usually distinctive, and here again the

* *Brain*, Part cxi.

electrical reactions will assist in the diagnosis. In very rare instances the diphtheria may cause permanent palsy of a bulbar type, cases of which have been recorded by Wilfred Harris.* The distribution of the paralysis in these cases very closely resembles that met with in myasthenia gravis, but, as Harris points out, the affection is usually limited to one or more of the bulbar nuclei, so that ptosis, weakness of muscles of the jaw and of the neck and limbs, are not present, as they so often are in myasthenia. Moreover, there will be no variation of symptoms and no myasthenic reaction, while, on the other hand, there will be wasting and reaction of degeneration.

3. *Spinal type*.—Where vague symptoms of spinal distribution are complained of, such as fatigue after slight exertion, an erroneous diagnosis of *neurasthenia* may easily be made.

Prognosis.—The outlook in the majority of cases is, in the long run, bad, though remissions and intermissions of long duration may occur, and in a small percentage of cases the symptoms seem to have permanently disappeared. The tendency is for the patient to become more and more easily exhausted, and the danger to life lies chiefly in the occurrence of attacks of cardiac failure and respiratory dyspnœa.

Treatment.—In absence of any definite knowledge of the cause of the trouble, efforts have to be directed towards preserving the contractility of the muscles. Rest, with the administration of strychnine and other tonics, is at present the best that can be done. The frequency with which an enlarged thymus gland has been found suggests the possibility of some modification or absence of an internal secretion as the cause of the disease, but hitherto the administration of glandular preparations has not been followed by any definite results.

* *Brain*, Part civ.

CHAPTER XXXVII

CONVULSIONS

It frequently happens that a patient is seen for the first time on account of "fits." It is therefore important to review the general subject of convulsions, which outwardly may give no clue to their different causes.

It is convenient to divide the subject into two parts, viz., convulsions as they occur (1) in *childhood* and (2) in *adults*.

1. **Convulsions in childhood.**—Some of the principal conditions which are associated with convulsions during childhood are: rickets, idiopathic epilepsy, infantile hemiplegia and diplegia from various causes, meningitis, and cerebral tumours.

The commonest condition which underlies the convulsions of children is rickets.

The well-known tendency to convulsions in children is considered to be primarily due to an incomplete development of the central nervous system, so that the different parts are not under adequate control. Thus a healthy infant is always more liable to convulsions than a healthy adult, but should, nevertheless, if properly cared for, possess a nervous system sufficiently stable to enable him to pass safely through this period of life, unless exposed to some unusually severe strain, as, for example, the incidence of an acute specific fever. If, however, the naturally incomplete development is further retarded by neglect,

rickets, or other circumstances, the stability will be still further lowered in varying degrees, and in that case comparatively slight variations in general health—intestinal disturbances, or other forms of irritation—suffice to bring on a fit.

Three types of children can be recognised in this respect:

(1) The healthy infant, in whom the relationship between different parts of the nervous system has not yet fully developed, but who still possesses a nervous system sufficiently stable under all ordinary circumstances.

(2) The child whose development has been abnormally retarded. Here, with care, convulsions may just be avoided, but the nervous system in this case is very lightly balanced, and a fit will readily occur from local or general disturbances.

(3) The child whose nervous system is so unstable that convulsions occur in spite of all care to avoid undue stimulation.

As the stability of the nervous system increases with the growth of the child the convulsions often cease, but in a certain number of cases this desired stability is never attained, and the child continues to be "subject to fits." It is this class which forms an important link between the convulsions of childhood and the epilepsy of adults, and between the two every gradation may be traced.

A recognition of these groups makes the part played by teething, constipation, flatulence, and other kinds of irritation in the causation of the fits easily understood. A healthy child may pass successfully through them all, but they will prove too strong for the possessor of an abnormally unstable nervous system and will start the impulses which give rise to the fit.

Dr. Eustace Smith has drawn attention to the possible occurrence of reflex convulsions in growing boys and girls of highly-strung and

excitable natures. The attacks are often preceded by premonitory symptoms of slight ill-health for several days, and in this way no doubt the balance of their nervous system, always finely adjusted, is overthrown. Dr. Eustace Smith lays stress on the frequent recovery of such cases and deprecates classifying them straight away under the head of "epilepsy."

The convulsions connected with the other cerebral conditions mentioned above are incidents in the courses of the diseases and do not require separate description.

2. **Convulsions of adults** are chiefly associated with epilepsy, toxæmias (e.g., uræmia, chronic alcoholism, and various poisons, such as strychnine), degenerations (e.g., in general paralysis of the insane), meningeal inflammations, cerebral tumours, hysteria, and onset of vascular lesions (hæmorrhages, thrombosis, embolism).

Reference to the chapter on Epilepsy (Chapter XXXIX.) shows that first attacks of this disorder become rarer as age advances, and, therefore, when convulsions occur for the first time after middle life, suspicions should be entertained of the possibility of cerebral degeneration (especially general paralysis of the insane), cerebral tumours, and uræmia. It must, however, be remembered that patients who have taken a great deal of alcohol sometimes have epileptiform fits about this age, which do not necessarily seem to signify the onset of any immediate cerebral degeneration.

In tumours of the brain a general convulsion, quite apart from the localised Jacksonian fit, is sometimes an early symptom, and may for a time be regarded as idiopathic epilepsy. It is therefore important to examine the discs of all patients who complain of fits of recent origin, even though it appears from absence of other symptoms that the fits are due to epilepsy.

The fits of the general paralytic may likewise be an early symptom of that disease, although they more often occur when the nature of the disease can be recognised from other signs. As far as the actual attack is concerned, it resembles that of an epileptic fit, either generalised or of a localised "Jacksonian" character, but whereas the ordinary epileptic generally shows no particular after-effects from the attacks, every fit tends to leave the general paralytic worse, both mentally and physically, than he was before.

Sometimes apoplectiform fits, closely resembling those of cerebral hæmorrhage, alternate with the epileptiform seizures, which does not happen in ordinary epilepsy. It is thought that the fits in these cases are induced by the irritative properties of the products of the degeneration of nervous tissue taking place in the cerebral cortex.

Uræmic convulsions occur occasionally as isolated symptoms, but are generally accompanied by definite signs of advanced renal disease. Sometimes they resemble cerebral hæmorrhage more than epilepsy.

Convulsions may occur in the initial stages of vascular lesions of the brain (hæmorrhage, thrombosis, and embolism), but the appearance of paralysis generally makes their origin clear.

Hysterical fits are described elsewhere (*see* p. 426).

CHAPTER XXXVIII

TETANY

TETANY is characterised by spasmodic contractions of the muscles, chiefly those of the extremities, and by an increase in the irritability of the muscles to electrical and mechanical stimulation.

Etiology.—Tetany is most commonly seen in children who are the subjects of rickets. More rarely it occurs in adults, and has been noted in association with chronic dilatation of the stomach, diarrhoea, debility after lactation, and removal of the thyroid gland. Occasional epidemics have been recorded.

Pathology.—The contractions are probably set up by a toxæmia which arises at some time during the course of the diseases mentioned above. No definite lesions have been found after death, and the exact seat of the irritative process, whether in brain, cord, or nerves, is a point which still remains undecided.

Symptoms.—The spasms, which are often preceded by sensations of tingling and numbness, are generally most prominent in the extremities. The hand usually assumes an attitude which is characteristic. The fingers are flexed at the metacarpo-phalangeal joints and extended beyond a position which is brought about by a spasm of the interossei, while the thumb is brought into the position of opposition and pressed tightly into the palm. At other times the fingers assume the shape of the “claw-hand.” The wrist and elbow

joints are flexed, and the arm is adducted. Tonic spasms of a corresponding nature occur in the feet, and the foot is inverted and the toes are flexed. In severe cases the spasms spread to the trunk and cause opisthotonos, and in some instances the tongue and muscles of the face are affected.

The attacks last for variable times. Sometimes the spasms pass off in a few minutes, and at other times they are more or less continuous for some hours.

Trousseau observed that an attack may be brought on by pressure on the main nerves and vessels of the limb.

The muscles and their motor nerves are very irritable to mechanical stimulation (Chevostek's sign), a fact which can be well observed in connection with the muscles of the face. The sensory nerves also are unduly irritable.

The excitability of the nerves to electrical stimulation, especially to that of the galvanic current, is greater than normal, and the anodal opening contraction is greater than the kathodal closure contraction.

Diagnosis.—The carpo-pedal contractions of a typical case are not likely to be confused with anything else.

When the spasms extend to the trunk and face, the possibility of tetanus may present itself, but is negatived by the order in which the contractions have appeared, as well as by the absence of trismus, which is such a characteristic early sign of tetanus.

Prognosis.—The occurrence of tetany in children is not usually dangerous, but sudden death has occasionally taken place in association with the spasms. As a complication of chronic dilatation of the stomach, tetany may be very troublesome or even dangerous; and if organic disease of the stomach be present, the onset of the spasms

may indicate the immediate necessity of obtaining some relief through a surgical operation.

Treatment.—In children the bowels should be relieved by castor oil, and enemata if necessary. Warm baths may be useful during an attack. Among the sedatives that are most likely to bring relief are bromides and chloral. Attention must be paid to the general health, which in the case of children is generally impaired by rickets.

biology

Children

Adults

Rickets

Chronic

Diarrhoea

Excision

Sebelly after lactation

Acidosis

Thyroid

CHAPTER XXXIX

EPILEPSY

EPILEPSY is characterised by a sudden and uncontrolled discharge from the nervous elements of the cerebral cortex. Varying in its intensity, the discharge gives rise to all degrees of disturbance, from a momentary loss of consciousness or a transitory attack of giddiness to a severe convulsion.

Etiology.—Little is definitely known about the causation of epilepsy. A consideration of convulsions in general seems to indicate that there are probably different groups of cases. In some it may be that a poison manufactured within the body is potent enough to cause convulsions (*e.g.*, those associated with uræmia); in others the poison may come from without. Thus it is not uncommon to see an elderly man have a convulsion, indistinguishable from epilepsy, as the result of chronic alcoholism.

On the other hand, there may be some local disease of the nervous system, and, as a result of this, toxins or other stimuli which are harmless to the ordinary individual prove sufficiently powerful to originate the discharge. It is almost certain that there is a local fault in many of those cases where the discharge, as is shown by the constancy of the warning, always begins in the same area of the cortex. Evidence of local origin is also seen where general epileptic fits are associated with organic disease of the brain, as when they follow an injury received at birth or the occurrence of a

cortical vascular disturbance during the first few years of life. Indeed, an injury to the brain at any age may be followed by general epilepsy.

In most cases the attacks first occur during youth; and it becomes less and less common to find them beginning from the age of 20 onwards, and when they have apparently done so a careful inquiry will often reveal the previous existence of minor attacks which have passed unnoticed.

Convulsions during infancy, whatever be their cause, dispose to epilepsy in later life, but there is often a considerable interval of freedom. Especial care, therefore, should be taken of children about the age of puberty, if they are known to have suffered from convulsions in infancy.

Epilepsy tends to run in neurotic families, in many of which there are also some members mentally afflicted.

When a nervous system is delicately balanced, any undue peripheral irritation may turn the scale, and, in this way, errors of refraction, nasal polypi, adenoids, ovarian and uterine disease, and other sources of irritation may, under certain circumstances, serve to start the explosion. In women there is sometimes some relation between the attacks and the menstrual periods.

At present our knowledge may be summed up in the statement that an epileptic fit depends upon some want of relation between the nerve cells of the cortex and their surroundings. In some cases, probably in many, it is the nerve cells that are at fault, so that toxins and stimuli are capable of producing a discharge quite disproportionate to their intensity, while in others the nerve cells may be of average stability, but the toxins reaching them may be too powerful to be withstood, just as the strongest of them will succumb to an overdose of strychnine or the poison of tetanus.

Symptoms.—Whether the cause is one of

local or general origin, the main characteristic of an epileptic attack is a nervous discharge from the cells of the cerebral cortex. Hence the symptoms vary according to (1) the part in which the discharge originates, (2) the direction in which it extends, and (3) the rapidity with which it spreads.

If a general view of the cerebral cortex is taken, it will be understood how the warnings occur in some cases and not in others: how in some the discharge is limited to a restricted area, and finally how in others a generalised convulsion results.

When the discharge starts in any of the areas the functions of which are known, its first effects are those of stimulation of the given area. Thus a discharge beginning in the *occipital lobe* will give rise to visual symptoms, and in the *temporo-sphenoidal lobe* to auditory symptoms, and so on. If the fit begins in the *motor area*, twitching of a limb is often the first sign, just as it is when a gross lesion causes the irritation.

Thus the nature and position of the aura, which may be referred to almost any part of the body, indicate the part of the brain at which the discharge commences.

The different parts of a fit are :

(1) Aura.

(2) Convulsions:

(a) Tonic.

(b) Clonic.

(3) After-effects:

Sleep, headache, and aching of limbs.

Coma.

Automatism.

Temporary mania.

Temporary paralysis.

At the termination or in the absence of the aura, the patient suddenly loses consciousness, and with a violent respiratory spasm which produces a groan or cry (the epileptic cry), falls to the

ground with the muscles in a tonic spasm. In about half a minute this stage is succeeded by clonic contractions, which last for two or three minutes and then gradually pass off, leaving the patient usually in a deep sleep, or leading to one or other of the post-epileptic conditions mentioned above.

The commonest *after-effect* is sleep of varying length, from which the patient is apt to wake with tired and aching limbs, as though he had been bruised all over. Sometimes the sleep deepens into coma.

THE AURÆ

These may be classified as sensory and motor.

1. **Sensory aura.**

(a) Special senses :

Visual.

Auditory.

Smell.

Taste.

(b) General, *e.g.*, sensations of something passing up a limb.

Tingling.

Giddiness.

Palpitation.

Flatulence.

(c) *Psychical, e.g.*, dreamy states, reminiscences.

Auræ of special senses.—Of these the visual and auditory are common, but warnings of taste and smell are rare. Visual auræ often take the form of flashes of light, partial or complete loss of vision, and images of various kinds, while loud noises and whistlings are most frequently met with in connection with the auditory warnings. Sometimes words are heard, or a part of a sentence which may always stop abruptly at the same place in successive attacks, when consciousness is lost. Warnings of taste and smell seem always to be unpleasant; an indescribable "sulphurous" smell

or a taste of "rotten cabbage" are among those described.

Auræ of general sensations.—The best-known is the sensation of "something passing up the arm or leg," from which the term "aura" was originally derived. The feeling generally begins in the hand and passes deliberately up the arm until it reaches a certain point, when consciousness is lost. With this kind of aura there is a good chance of arresting the fit by tying a cord round the arm and pulling it tight the moment the sensation is felt—a practical device which has been handed down from ancient times.

Most of these general sensations are vague and call for no special description, and in many of them the patients find it difficult to express their feelings in words.

Auræ of psychological sensations.—These warnings are interesting, and include various dreamy conditions which are often not altogether unpleasant. There also occur the peculiar sensations known as "reminiscences," in which there is a feeling of familiarity with a scene, as though it had all been seen before, though it is often impossible that there can have been any previous knowledge of it. These sensations are experienced by healthy people, and have been well described by Dickens, Scott, Tennyson, Oliver Wendell Holmes, and other writers; but when they occur very frequently and vividly they should be regarded with suspicion, and careful inquiry should be made for any other symptoms, such as attacks of momentary dizziness, or the signs of spasm in the jaws, face, or eye muscles, which, if present, will clear the way for commencing treatment.

—As a rule the aura lasts from a few seconds up to a minute or so, but sometimes it is of considerably longer duration, and enables the patient to try to check the fit and to get into a place of safety.

In about half the cases there is no aura at all.

Indefinite feelings, headaches, depression, and irritability sometimes enable the patient to prophesy the onset of a fit some hours, or even a day or two, before its occurrence.

2. **Motor aura.** — This consists of twitching of parts of limbs, and where the extension of the discharge proceeds slowly its progress can be traced by the movements of successive parts of the limbs, just as may be done when a tumour causes the fit (Jacksonian epilepsy). It is important to emphasise the fact that the characteristics of Jacksonian epilepsy, where the convulsion spreads slowly from part to part, and either remains local, or becomes general with loss of consciousness, are by no means confined to cases where there is a tumour or other gross lesion (in which connection the term was first used), but are very often found in association with attacks of "idiopathic" epilepsy.

Post-epileptic automatism. — Here the patient performs co-ordinated acts without any knowledge of what he is doing: thus he may walk for hours, and on coming to his senses may find himself miles from where he intended to go; or he may drive through crowded cities without any remembrance; and, most important of all, he may commit any kind of crime while he is in this irresponsible condition.

The important point in this connection is that the automatism is just as likely, or perhaps more likely, to follow a minor fit which may be so slight as to pass unrecognised by those around, who therefore only see in the illegal act the impulse of the criminal. Mental disturbances sometimes appear to take the place of a fit, and are then spoken of as "psychic equivalents."

Post-epileptic mania is not very common. It is often of temporary duration, but while it is present the patient may be very violent.

Post-epileptic paralysis. — After a fit, one or

more limbs are sometimes paralysed. The cause is presumably nervous exhaustion, and the power generally quickly returns. It is not a common occurrence.

Details of a fit.—It will now be instructive to follow out a fit in detail.

If the patient be subject to a visual aura, the discharge will begin in the occipital area which is the seat of the function of vision, and the patient has the visual sensations which may take the form of bright lights or dimness of vision. The face will probably become pale.

The discharge, having once broken down the surrounding resistance, spreads with lightning rapidity over the motor and frontal regions. The part of the motor area which generally appears to feel the effect first is that connected with lateral movements of the head and eyes, and hence the head and eyes turn to one or other side (conjugate deviation). Almost synchronously with this movement the discharge spreads over the rest of the fore part of the brain. Consciousness is lost, and the intense stimulation of the motor area throws the whole body into a sustained spasm. The patient becomes quite rigid and falls like a log, uttering at the same time a cry, groan, or gurgling noise. He is now in the stage of tonic spasm, in which the muscles of respiration share, and consequently the face becomes turgid and the veins swell. Frothy fluid appears at the lips. After a few seconds a little twitching is seen, beginning first probably in the face and then gradually becoming general; the limbs are alternately violently flexed and extended; the champing jaw often catches the protruding tongue, and blood is added to the frothy mucus in the mouth. The sphincters are frequently relaxed.

The movements gradually diminish, until in two or three minutes all is quiet again, and the patient either gets up and goes about his business

in a more or less dazed condition, or, if the circumstances are favourable, sleeps for several hours.

The conjunctival reflex is lost throughout the fit, and the pupils are dilated and do not react to light.

Immediately after a severe fit the knee-jerks may be temporarily abolished owing to the exhaustion of the pyramidal fibres consequent on the convulsion, but more often the knee-jerks are increased, and ankle clonus and temporary extensor response of the plantar reflex may be present.

Status epilepticus.—In the status epilepticus, which is a rare complication of epilepsy, the patient has a rapid succession of fits, and in the more severe forms there are no intervals of consciousness between the attacks. It is always a serious condition, and in many cases death occurs from exhaustion.

Minor epilepsy.—Minor attacks are variously designated as “faints,” “turns,” and “dizzy” and “peculiar” feelings. Apparently they are due to localised discharges of momentary duration, and their signs therefore pass off rapidly and are very limited in their distribution. The most common variety is characterised by a momentary loss of consciousness. The patient “loses himself” for a few seconds, stops abruptly in his conversation or work, perhaps drops something, and then takes up the thread again and continues as before. Such attacks often pass unnoticed, but, if attention is drawn to them, a passing pallor of the face, with possibly a slight twitching of some muscles, may be observed.

The “reminiscences” to which allusion has already been made are also common in minor epilepsy.

Visceral, auditory, and visual sensations are all common, and indeed any of the sensations which have been described as the aura of the major

fits may occur as manifestations of the minor variety.

In this way the minor attacks may be looked upon as abortive fits in which the discharge remains strictly circumscribed. Presumably these slight discharges are particularly apt to arise in the frontal region, since momentary loss of consciousness is such a common event. A patient may suffer from minor attacks for years before a major fit occurs; in other cases both frequently occur, but in many the major attacks alone occur.

Post-epileptic automatism, as already remarked, is very liable to follow a minor fit which may have been so slight as to pass unnoticed.

Diagnosis.—Epilepsy has to be distinguished from convulsions which have a definite organic or toxæmic basis.

From convulsions of infancy.—Little children are, as we have already seen, very liable to convulsions, which in the majority of cases cease to recur as development takes place. The individual convulsions are indistinguishable from those of epilepsy, but, bearing in mind this special tendency of childhood, it is permissible to hope that in any given case the fits will pass off as the child grows older. Suspensions may be aroused by the history of other members of the family, and by the fits occurring in an apparently quite healthy child without any adequate associated conditions such as rickets.

Lastly, it must be remembered that a number of convulsions in childhood undoubtedly make for the occurrence of fits in later life, and it is common to find a recurrence of the convulsions about the age of puberty, even though there have been several intervening years of freedom.

From convulsions due to organic disease in children.—If the organic disease is acute, e.g., meningitis, the convulsions are, of course, for the moment, a symptom of minor importance. If the

fits are associated with any of the forms of infantile hemiplegia or diplegia, or follow as a late result of meningitis, from hydrocephalus, then they are likely to assume the ordinary character of epilepsy.

From *cerebral tumour*.—There are two conditions in which epilepsy may be confused with cerebral tumour:

1. *When the convulsion is general*.—Occasionally it happens that a general convulsion, indistinguishable from an epileptic fit, is the first sign of a cerebral tumour. While, of course, bearing in mind the possibility of other symptoms, the most certain way to avoid mistaking the generalised convulsion of a tumour for epilepsy is by examining systematically the optic discs. It is very likely that the appearance of the fundus will be suspicious even if there are no other symptoms to suggest the true nature of the disease.

2. *When the convulsion is local*.—Tumours situated in the motor area of the brain cortex cause fits, which begin by localised movements that deliberately progress to other parts of the limb, and may or may not terminate in a general convulsion with loss of consciousness.

It must be remembered that many cases of ordinary epilepsy also begin in this way, and that in the complete absence of all other symptoms of a tumour the case is likely to be one of "idiopathic" epilepsy.

From convulsions due to *general paralysis*.—General paralytics are always liable to convulsions, and the possibility of these must always be remembered when a middle-aged man or woman suddenly has a fit for the first time.

From *uræmic convulsions*.—Uræmia sometimes first shows itself by a convulsion, but an examination of the urine will usually make the nature of the case clear.

From *alcoholism*.—Chronic alcoholics some-

times have for the first time in middle life a fit which appears to be epilepsy, though in many it is probable that the effects of renal disease are complicating factors.

From hysteria. — The difficulty of deciding whether a fit is hysterical is chiefly due to the fact that in most cases the medical man has only the history to depend upon.

The chief points on which reliance is placed in making the differentiation are as follows:

	EPILEPSY.	HYSTERIA.
Cry ...	A single cry or groan...	Often several shrieks.
Fall ...	Sudden, <u>without any attempt to save themselves</u>	<u>Slide down</u> ; seldom hurt themselves by falling.
Sphincters	<u>Often relaxed</u> ...	Not relaxed.
Tongue ...	<u>Bitten</u> ...	Not bitten, but lips may be.
Struggling	<u>No purposive struggling</u>	Often struggle and fight, requiring several people to hold them.
Presence of others	Fits occur <u>when patient is alone or in presence of others</u>	Usually in <u>presence of others</u> .
Duration ...	A few minutes ...	<u>Often a long time</u> .

It is most necessary to remember that an hysterical fit may directly succeed one of epilepsy, and that the latter may be of the "minor" kind, and be quite unnoticed by the ordinary bystander.

Prognosis.—It is difficult to estimate with any accuracy the number of cases in which fits have entirely ceased, for such patients naturally escape further observation. It is generally thought that the disease is arrested in from 5 to 10 per cent. of all cases. It is impossible to predict what course the disease is likely to take until the effects of steady treatment are known, for it is very rare indeed for the fits to disappear spontaneously.

Long intervals between fits, and absence of minor attacks, are both favourable, and cases beginning in later life are generally more easily influenced than those arising about the period of puberty. Statistics show that the prognosis is no worse in cases where there is an hereditary tendency. The danger to life, except when the rare condition of status epilepticus occurs, arises from possible accidents, of which the chief are suffocation from turning on to the face in nocturnal epilepsy, injuries or death from a fall when consciousness is lost, and choking from the passage of vomited food into the larynx. In a considerable number of confirmed epileptics there is mental deterioration, which not unfrequently becomes advanced enough to constitute insanity.

Treatment.—To treat epilepsy with any degree of success the utmost attention must be given to details.

The daily life should be arranged to avoid any conditions which are likely to cause sudden variations in the equilibrium of the nervous system.

Peripheral stimuli of undue strength from without, as the sudden appearance of bright lights, loud noises, and other things causing "starts," must be avoided just as much as those from within, and a quiet, even life, with perfectly regular hours, should be led. The occupation should be physical and mental, judiciously proportioned to each case. It is most important not to allow idling and day-dreaming. All evidence shows that occupation reasonable in kind and degree is beneficial, and it is largely due to the possibility of leading a mechanical life, with proper occupation, that epileptic colonies are so successful. It is not common for epileptics to have fits when they are engaged in some engrossing occupation; the dangerous moments seem rather to be those in which the mind is aimlessly

wandering, and just between the sleeping and the waking condition is a favourite time for a fit.

Arrest of fits at their onset.—Many patients find out for themselves that they can frequently ward off a threatening fit by “force of will.” This power should be encouraged, and it may often be aided by muscular exertion. The moment a fit threatens, the patient should clench his teeth and grasp something tightly. The most convenient method is to clasp his own arm as tightly as possible, and in many instances the discharge can in this way be arrested.

The possibility of arresting fits by tightening a cord when the aura passes up a limb has already been mentioned, and, when practicable, should always be employed. Smelling strong salts and inhalations of amyl nitrite sometimes meet with success. Every fit stopped is a distinct gain, and where the fits are few and far between it is most important to make the gaps as wide as possible.

Diet.—The food should be simple and quite plainly cooked, and spices of all kinds should be avoided. The question of allowing meat is one on which there is some difference of opinion. Certainly, the meat taken should be moderate in amount and of an easily digested nature, but there does not seem any great benefit in abolishing it altogether, at least in the majority of cases. More recently it has been thought that the harmful constituents of food are the “purin” bodies,* and diets have been constructed to eliminate these. Such a diet would include milk, eggs, butter, cheese, rice, macaroni, tapioca, white bread, cabbage, lettuce, sugar, and fruit, while in potatoes and onions the amount of purin bodies is very small. Tea, cocoa, coffee, meat, and fish

* The purin bodies are substances of which the composition C_5N_4 forms the basis; they are found in many common foods, and also as the result of tissue metabolism of the body. See “Purin Bodies of Food-Stuffs,” by Walker Hall.

should, on this hypothesis, be avoided. Among the meats which contain the least quantities of purin bodies are tripe, neck of pork, and cod-fish.

Aldren Turner* has found that diets constructed on this principle seem to be beneficial in some instances, but, like other forms of treatment, are not likely to produce an effect that is constant for all cases.

Elimination of salt.—The elimination of salt from the diet has been advised in recent years on the supposition that the bromide can take its place in the tissues of the brain. By this means it was hoped that the fits might be controlled by smaller doses of bromides, which at the same time would have a more powerful effect. No constant results have been obtained, but a certain proportion of cases seem to have benefited, so that it is often worth a trial.

Drink.—Alcohol should be absolutely forbidden. Coffee is too stimulating; but weak, fresh tea may generally be taken. Water and milk are the best drinks for general purposes.

Bowels.—The bowels should be most carefully regulated. Constipation very often appears to be the exciting cause of an attack.

Drugs.—The bromides of potassium, sodium, and ammonia, given judiciously, are the best drugs in epilepsy, and, in spite of its more depressing influence, potassium bromide is the one which appears on the whole to give most satisfaction. Many prefer to give the different bromides together. Bromide of strontium also is useful.

Regulation of dose.—Much of the success in treatment depends upon carefully regulating the doses. If too little is given, the fits are not checked; while if the amount is unnecessarily large, mental depression and dulness are produced.

* *Practitioner*, April, 1906.

The ideal dose is that which is just sufficient to check the fits.

It is, of course, impossible to lay down any rules that are applicable to all cases, but in the case, say, of a young man with a history of only one or two attacks at long intervals, it is most important that those attacks should be arrested or be limited to very long intervals, and there is a reasonable probability at such a stage of at least attaining the second of these objects. Every fit he has in the future will make this probability less, hence it is better to err on the side of plenty in order to make sure of checking the fits, even at the risk of causing some temporary depression.

On the other hand, in a confirmed epileptic there is not the same urgency, and the suitable dose can be more deliberately arrived at. Broadly speaking, the dose limits of bromide in epilepsy are between 10 and 80 grains a day. Larger doses are sometimes needed, but as a rule are not beneficial when the smaller amount has no effect.

Time for giving bromides.—Much may be done by considering the times at which fits usually occur in relation with the taking of medicines. Where the attacks show no regularity the doses may be taken at convenient intervals twice or three times a day as required, but where it is possible to calculate to some extent on the onset of a fit, a judicious anticipation will often meet with success. Thus if the fits occur only during sleep or in early morning, a single dose of bromide of 20 or 30 grains may be taken the last thing at night.

Drawbacks of bromide.—The drawbacks to taking bromides continuously are the tendency to the production of:

(1) Acne.

(2) Mental depression.

The tendency to acne can be counteracted by combining small doses of arsenic with the bromide.

This in its turn has a drawback, for arsenic taken over a long period of time may give rise to pigmentation of the skin. When combined with bromide the arsenic in the doses usually given never appears to produce neuritis or other unpleasant effects which it is sometimes apt to do when taken in other ways.

An example of a prescription is as follows:

R. Potassii bromidi, gr. xx.
Liquor arsenicalis, m. iii.
Spiritus chloroformi, m. xv.
 Aq. ad ℥i.

Two tablespoonfuls to be taken twice or three times a day
 after food.

As already mentioned, the bromides of sodium or ammonia may be substituted for that of potassium, or the dose may be divided between the three.

Adjuvants to bromides.—While bromides form the basis of nearly every prescription for epilepsy, there are other drugs which, when combined with them, appear often to have some good influence. Of these the most important are belladonna and borax, either or both of which may be combined with the bromides, e.g.:

R. Potassii bromidi, gr. xx.
Sodii biborate, gr. v.
Tinct. belladonnæ, m. v.
Liquor arsenicalis, m. iii.
Sp. chloroformi, m. xv.
 Aq. ad ℥i.

Two tablespoonfuls to be taken three times a day.

The ordinary major fits which are not checked by various doses of some such mixture as the above are not likely to be much lessened in frequency or severity by anything else, though occasionally the addition of small doses of digitalis and potassium iodide seems to have a good effect. Iron may be of assistance when there is anæmia, but in some cases it disagrees. Nitro-

glycerine may be given with the bromides when the condition of the peripheral circulation seems to warrant the administration of vaso-dilators.

The minor fits are often more difficult to control than the major ones, and where bromides fail, pills of zinc oxide may be tried. An endless number of drugs has naturally been tried and commended at various times, but no mixture meets with such uniform success as that of which bromides constitute the basis.

Surgical treatment, so useful where fits depend upon a gross lesion, is very seldom indeed of use in general epilepsy. Here and there a very local and deliberate onset may suggest the advisability of removing a piece of the cortex, but in the large majority of cases no operation is likely to produce a cure.

Summary of treatment.—The points to which attention should chiefly be given may be summed up as follows:

- (1) General mode of living.
- (2) Diet.
- (3) Alimentary canal.
- (4) Removal of all sources of peripheral irritation.
- (5) Administration of drugs, the chief of which are bromides.

Summary of Treatment.

1. General mode of living.
2. Diet.
3. Alimentary Canal
4. Removal of all sources of Peripheral Irritation
5. Drugs. chiefly Bromides.

CHAPTER XL

PARALYSIS AGITANS (PARKINSON'S DISEASE)

PARALYSIS agitans is characterised by the gradual onset of weakness, tremors, and rigidity.

Some degree of weakness is probably present in all cases, but the amount of tremor and rigidity is very variable.

From the point of view of diagnosis, rigidity is quite as important a symptom as tremor, and the nature of the disease is often clear though there may be no tremor.

Etiology.—It is not possible to attribute the onset to any direct cause, though injury, shock, worry, or anything that causes general debility sometimes appears to be the starting-point. Between 45 and 60 is the age at which the disease usually begins, and men are rather more commonly affected than women.

Pathology.—Nothing definite is known concerning the underlying changes in the nervous system. Alterations in the structure of the cortical cell bodies and their dendrons have been found by some, while others have described arterial and perivascular changes; but none of these seems to be constantly present. It has recently been suggested that the modification or want of some internal secretion, more especially of that connected with the parathyroids, may be the cause. Others consider that the essential changes take place in the muscles.

Symptoms.—The onset is usually insidious.

Occasionally the tremor seems to date definitely from the time of an injury or shock, but more often it begins so gradually as to be scarcely noticeable.

In some a slow, progressive weakness, with increasing stiffness, marks the onset; while in others the weakness, tremors, and rigidity all appear at about the same time.



Fig. 88.—Writing in a case of paralysis agitans.

The tremors are generally first noticed in one hand; the thumb and forefinger move rhythmically in a way which has been aptly described as "pill-rolling" in character. At first the trembling is scarcely noticeable, but gradually obtrudes itself until it begins to interfere with ordinary actions.

From the forefinger and thumb the tremor



Fig. 89.—Line drawn by a patient with a severe tremor, showing power temporarily to control the tremor.

spreads to the other fingers, and at last the whole hand takes part in the movement, which later may extend to the elbow and to some extent to the shoulder.

By an effort of will the movements can be controlled, but only for a few seconds, and in severe cases an attempt to keep one limb still generally results in increased movements of another.

The tremors are most obvious when the limbs are at rest, and, indeed, during brief voluntary



Fig. 90.—Case of paralysis agitans showing the characteristic attitude.

movements, *e.g.*, holding out the hand, they become distinctly less, though they do not altogether cease, as is sometimes stated.

This remission during voluntary movement

can be easily demonstrated by asking the patient to write or draw a line, an action which, from the tremor present, would often appear almost impossible, but which on trial often proves to be done comparatively well (Fig. 89).



Fig. 91.—Hyperextension of the thumb in a case of paralysis agitans.

The tremors cease during sleep.

From one hand the tremor generally spreads to the leg on the same side, and thence to the hand on the other side, but there are many exceptions to this rule. Some of these are more

apparent than real, for the tremor in the legs is often less noticeable than that in the arms, probably because the muscles are more in action, for, as already mentioned, movement becomes more obvious when the muscles are relaxed.

As the disease progresses, the head, face, and jaws may all take part in the movements.

Rigidity is even more characteristic of paralysis agitans than tremor.

At first scarcely noticeable, it creeps on relentlessly until the figure becomes firmly set in the characteristic position (Fig. 90). Obeying the usual laws, we find the flexors overbalancing the extensors, and the patient becomes bowed like an old man, with the head bent forward and the body flexed. The arms are abducted at the shoulder and flexed at the elbows, while the fingers and thumb are bent into a half-closed position. The thumb is often curved in a position of hyper-extension (Fig. 91).

The features are fixed and expressionless (Parkinson's mask). The voice is dull and monotonous, owing chiefly to the rigidity of the muscles of the lips and face. It can easily be imitated by trying to talk without moving the lips.

Voluntary movements are performed slowly and stiffly, and the relaxation of muscles takes place more tardily than usual.

The rigidity of the bowed body with the upper part bent over throws the centre of gravity forward and leads to the peculiar gait, in which the patient takes short, rapid steps that oftentimes amount to a run, and when once started he has considerable difficulty in stopping. From his appearance it has been well said that the patient looks as if he were trying to catch up his centre of gravity.

The mechanism of this peculiar gait can be best understood by imitating the conditions under

which it occurs. If the body is bent well over and kept stiff, so that the balance is only just preserved, and a short step forward is taken, there will be a tendency to fall, which is only counteracted at each step by rapidly shuffling the hind foot forward. This movement is repeated indefinitely, and the patient being unable to bend the body back, and so to restore the balance, there is difficulty in stopping without running the risk of falling, unless the patient catches hold of something for support.

In the same way, if the patient is suddenly started backwards, the rigidity of his body makes it difficult to regain the balance, and this is involuntarily continued for some steps. This is known as retropulsion. A similar effect can also be obtained by suddenly starting the patient on a side movement (lateropulsion).

The deep reflexes are generally said to be unaltered, but in many cases, especially before rigidity becomes marked, the author has found them increased. The plantar reflex is not altered.

Among other symptoms are cramps, which are often troublesome. Some patients complain of the toes drawing up under the feet and troubling them in walking.

Sensations of heat and cold are common, and are apparently of vasomotor origin.

In the later stages, when it is almost impossible to move voluntarily, there is often very great restlessness, with a desire to have the position of the limbs constantly altered.

Prognosis.—The disease progresses slowly, and finally the patient may be quite bedridden. There is no special danger to life.

Treatment.—The only chance of doing anything for these cases is to begin treatment early. Even then it is only palliative, but undoubtedly much comfort and some retardation of symptoms can often be obtained by judicious treatment.

This has to be directed towards relief of the three main symptoms: tremors, rigidity, and weakness.

The cases in which the tremors are severe are the most painful, for the patient is never able to be at rest, and, unfortunately, no drugs can be depended upon to still them. All the usual sedatives may be tried, but only seldom is any lasting effect produced. Opium and its preparations are of no use.

When the tremors are very bad, hyoscine sometimes checks them to some extent. It may be given by the mouth or hypodermically, but it is a drug which it is necessary to use with caution on account of its toxic effects.

In slight cases the patient should be encouraged to control the tremor, but in the more severe any sustained effort is very exhausting. Little devices of various kinds can sometimes be found to give comfort. In one patient who was under the author's care, carrying a heavy stick gave some relief to the tremor.

It is scarcely necessary to state that as quiet a life as possible should be led. Excitement makes the tremor worse, and in some instances, so long as the patient remains quiet, it gives him very little trouble for quite a number of years.

The rigidity is best combated by carefully regulated active and passive movements, and if these are practised early enough there is no doubt that the tendency to permanent fixation may be postponed. Hot baths regularly given and accompanied by light massage also give relief.

The weakness requires no especial form of treatment. The main point is to keep up the general nutrition; the diet should be suitable in quantity and quality; and the patient should live as much as possible in the open air, a mode of life which seems to exercise a general influence in retarding the progress of the disease.

CHAPTER XLI

CHOREA (RHEUMATIC CHOREA, SYDENHAM'S CHOREA, ST. VITUS'S DANCE)

CHOREA is characterised mainly by the presence of involuntary movements, together with varying degrees of muscular weakness and incoordination. Frequently there are also some mental changes present.

Etiology.—Chorea is a disease chiefly of children between the ages of five and fifteen, and occurs more often in girls than in boys.

Rheumatism is undoubtedly the principal underlying cause, and statistics show that a definite history of rheumatic manifestations can be obtained in a very large number of cases. Less often an attack of rheumatism follows chorea, and only occasionally are the two conditions associated.

Acute specific fevers other than rheumatism are not directly concerned in the causation of chorea. The apparent relationship of the disease to scarlet fever may depend upon the close association which exists between the latter affection and rheumatism.

In the rheumatic child, anything which strains the nervous system seems to predispose to an attack of chorea; hence inherited nervousness, too much schooling, frights, errors of refraction, and other forms of irritation often appear in the history of the case as the apparent causes. Fright is especially apt to be closely followed by chorea.

Chorea is sometimes associated with pregnancy,

but in the majority of these cases also some history of previous rheumatism can be obtained.

Pathology.—It is now generally conceded that this form of chorea is a manifestation of rheumatism. Poynton and Holmes* consider that the *Diplococcus rheumaticus*, the presence of which they have demonstrated in the pia mater of fatal cases, is probably the infective agent; but the exact nature of the infection is still a subject of controversy.

Among the microscopical changes that have been observed in the cerebral cortex are hyperæmia, thrombosis, swelling and chromatolysis of the cortical cells.

Symptoms.—The onset is often insidious, and begins with "inattention," "dropping things," and "fidgiting." Sometimes, especially after a severe emotional disturbance, the onset is acute, and either weakness or irregular movements appear quite suddenly.

The symptoms may be divided into the following groups:

1. Involuntary movements.
2. Muscular weakness.
3. Incoordination.
4. Mental changes.
5. Associated rheumatic manifestations.

1. *The movements.*—The movements are spasmodic, involuntary, and irregular. They may be distributed all over the body, or they may be confined to one limb or to one side (hemichorea). Their intensity varies in degree: in one case it may be quite impossible to keep the patient in an ordinary bed; in another the movements can only be detected with difficulty. In slight cases the movements generally become more obvious if the child holds out both hands with the fingers widely outspread, and at the same time protrudes the tongue.

* *Lancet*, Oct. 13, 1906.

Irregularity is a characteristic of the movements, and the same movement is never repeated continually, as it is in the tics. The hands are generally more severely affected than the upper arm, and when the fingers are spread out there is a tendency to hyperextension. Peculiar grimaces are made, which, together with spasms of the tongue and jaw, often make talking and eating difficult.

The diaphragm may contract spasmodically, and the heart may beat irregularly. Excitement of any kind makes the movements worse, and in severe cases the whole body is tossed about with great violence; but during sleep they disappear.

2. Loss of power.—There is probably some weakness in almost every case of rheumatic chorea, but it is often difficult to differentiate it from the spasmodic movements and incoordination, both of which cause apparent loss of power in the holding of objects.

Loss of power in one or more limbs is, however, an important feature in a considerable percentage of cases, and occasionally it is the predominating one, in which case the term "paretic chorea" is sometimes used. At first sight such cases may simulate the paresis of acute anterior poliomyelitis.

3. Incoordination.—There is considerable difficulty in carrying out voluntary movements with precision, as is easily seen by asking the child to perform some delicate act, such as picking up a tiny object, when the fingers move irregularly round it before it is finally seized.

There is no constant alteration in the reflexes. The knee-jerks are sometimes difficult to obtain, but when they are elicited they are often rather exaggerated. Sometimes, owing to the irregular contraction of the quadriceps extensor, the leg hangs in the air longer than usual, a condition which has been termed the "sustained knee-jerk."

The sphincters are unaffected.

4. Mental changes.—Dulness, want of attention, and inability to do lessons are often noticed. In bad cases, especially when it is difficult to feed the child, sleeplessness may be a troublesome symptom. In the severe cases there may be acute delirium accompanied by a high temperature.

There are no definite sensory changes peculiar to chorea. Occasionally there is some degree of anæsthesia of varied distribution, and the disease may sometimes be further complicated by the presence of hysterical manifestations. In some cases, too, complaint is made of pains in the limbs.

5. Associated rheumatic manifestations.—Endocarditis is the most important rheumatic manifestation that frequently accompanies chorea; the mitral valve is the one usually affected, and a systolic murmur can generally be heard at the apex. In fatal cases, bead-like vegetations of recent origin may be found upon the cusps.

Erythema nodosum may be present in conjunction with the chorea.

Joint troubles are more likely to have occurred at some previous time, but occasionally they are met with during the attack.

Diagnosis.—The irregularity and persistence of the movements serve to distinguish chorea from the tics. In the tics, periods of complete repose alternate with exacerbations (p. 396).

The athetosis of hemiplegica or diplegica in children is a much slower movement, and, moreover, the persistence of the movements and the permanent paralysis, which is frequently accompanied by arrested mental development and epilepsy, all point to the nature of the case. The history is also frequently an aid in diagnosis, for in most cases it is found that the paralysis (if not the athetosis) and the mental changes date from birth, or have supervened shortly after at an age at which chorea never occurs.

Hysterical movements may closely resemble chorea, but, like those of the tics, they are usually more regular. In hysteria, other confirmatory manifestations will usually be found, but it must not be forgotten that it is possible for both conditions to be present at the same time.

In myoclonus the spasms are shock-like. The trunk is more liable to be affected, and the contractions tend to be symmetrical.

Prognosis.—Patients with rheumatic chorea generally tend to get well in the course of a few weeks, but the progress is often slow, the affection lasting on an average between four and twelve weeks. There is a strong tendency for the patient to relapse just when the symptoms seem to be abating, and there is also a distinct tendency to recurrence after varying intervals.

Exceptions to this benign course of the disease occur every now and then. Severe cases in which the body is incessantly thrown about may end fatally with symptoms of exhaustion, delirium, and hyperpyrexia, and to such the term chorea gravis is applied.

Endocarditis which develops during the course of chorea has the same significance as that developing during the course of rheumatic fever and other diseases.

Treatment.—The main objects of treatment are :

1. To obtain rest (mental and physical).
2. To improve nutrition.
3. To give such drugs as may be thought to have a special influence on the disorder.

1. *Rest.*—In cases of any severity the child should be kept in bed. In others the time should be spent quietly, free from all excitement, and when possible it is desirable to avoid the society of other children.

2. *Nutrition* must be improved by a diet con-

taining plenty of milk; preparations of cod-liver oil, iron, phosphates, and other similar tonics are very useful.

3. *Drugs.*—The drug that has gained most popularity in the treatment of chorea is arsenic, and it is usually recommended to begin with doses of 2 or 3 min. of liquor arsenicalis three times a day, and gradually to increase the dose to 10 min. or more. While a careful watch will probably be kept for acute symptoms of arsenical poisoning, it must be clearly understood that there is also a very real danger of setting up arsenical neuritis when large doses are given for any length of time, and, further, the benefit to be obtained from this method of treatment is by no means assured. In any case, there seems to be no certainty that arsenic exercises any specific action on chorea.

Salicylates and aspirin are extolled by some, but, in spite of the close relationship of chorea to rheumatism, there is no consensus of opinion in favour of these drugs.

Phenacetin and antipyrin are also regarded as useful.

In acute cases, chloral hydrate and the bromides may be required to produce sleep.

As a general rule, rest, good food, with cod-liver oil and iron or some other suitable tonic, should form the basis of treatment, while arsenic, salicylates, phenacetin, or other drugs may be used additionally as occasion demands.

Besides treatment by drugs, the neurotic disposition of the child needs careful consideration, and the induction of happiness and contentment often materially assists the cure.

Dr. Leonard Guthrie has found that suggestion combined with passive movements, the performance of voluntary movements under guidance, and, in the later stages, correction of incoordination by means of graduated exercises, are all useful adjuncts to other methods of treatment.

HUNTINGTON'S CHOREA

This is an hereditary form of chorea, and bears no relation whatever to rheumatic chorea. It can often be traced through several generations; both sexes suffer, but it is said never to skip a generation, so that once the tendency is broken it does not reappear.

The symptoms usually begin after middle age, and consist of choreic movements and ataxy together with mental degeneration. The movements cease during rest, but are otherwise very persistent; they are slower than those of rheumatic chorea.

|| Ataxy is a prominent feature, so that there is often a staggering gait like that of a drunken man.

The mental changes are chiefly characterised by progressive enfeeblement.

The disease often lasts a number of years. No adequate treatment is known.

SENILE CHOREA

Occasionally in late middle life cases occur which resemble in many respects those of Huntington's chorea, but in these senile cases no other members of the family suffer. The prognosis is not good.

ELECTRICAL CHOREA

Electrical chorea is the term applied to a rare condition in which sudden movements like those from an electrical shock are associated with wasting of muscles and pyrexia. This disease, which progresses to a fatal termination, was first described by Dubini, and appears to occur in Italy only. The cause has been ascribed to some infective process.

CHAPTER XLII

THE TICS

THE tics are spasmodic movements of cortical origin which are repeated time after time, without any definite cause or object.

They represent voluntary movements which would be useful in their proper place, and their origin can often be traced to the repetition of movements which were at one time performed with a definite purpose. In this way persistent blinking may result from errors of refraction or injury to the eye; sniffing, from nasal affection; and shrugging the shoulders, from ill-fitting clothes. Normally, all such movements should cease when the causes which evoked them are removed, but in neurotic or debilitated people they may persist indefinitely. Several different movements may be out of hand at the same time, such as blinking the eyes and twitching the mouth muscles.

These tics must not be confused with the spasms accompanying neuralgia, which have received the name of "tic douloureux."

The varieties of tics are innumerable. When the movements are confined to a small group of muscles, *e.g.*, in blinking or sniffing, they are often called simple tics or habit spasms, while the more complex ones are designated as coordinate tics, though all true tics are, as a matter of fact, coordinate movements.

There are also mental tics, in which an idea or a tune constantly repeats itself, though here, again,



PLATE XIX.—Successive Stages of Stumbling Movements in a Peculiar Gait due to a Co-ordinated Tic.

it must be understood that there is a psychical element present in the production of all tics. Tics of speech also occur and may coexist with impulsive movements of the body; in some of these the patient repeats some words which he has just heard spoken (echolalia), and in others blasphemous or obscene expressions are habitually involuntarily uttered (coprolalia). Latah, or the jumping disease, found especially among Malays, is probably a complicated form of tic.

echolalia
coprolalia
Latah

Etiology.—Allusion has already been made to the origin of tics from movements which were at first performed with a definite object. Heredity and a neurotic temperament are both important conditions, as also is mimicry. Tics of slight severity often arise from overwork or general debility, and disappear again as the general health improves.

Character of the movements.—The movements constituting the tics usually consist of short, sharp spasms, which may be executed with great rapidity, and which have been compared to the movements from an electric shock; or they may be more deliberately performed with a rhythm that seems quite voluntary, and occasionally they assume a tonic character. The rapid, lightning-like jerks are generally well seen in the facial tics.

The performance of a tic is usually preceded by a desire to make the movement and followed by a sense of depression at having made it, but after a time the movement may take place unconsciously.

The spasms are apt to occur in bouts, with intermissions of varying length. Any attempt to control the movements involves great effort, which can only be sustained a very short time, and which is usually followed by a specially severe attack. The tics, as a general rule, disappear during sleep.

Diagnosis.—In children the movements are liable to be mistaken for those of *chorea*.

The distinction can be made by noting that

1. The same movement or movements are constantly repeated in tics, whereas in *chorea* the movements are irregular and various.

2. The movements in tics have a more distinct, purposeful character, though they have no adequate cause and bear no definite result. Those of *chorea* are irregular and in no way suggestive of purpose.

3. In the tics there are definite bouts, with intervals of complete freedom. In *chorea* there are exacerbations, but the intervals are occupied by movements, even though slight, so that there is not the complete intermission that is present in the tics. —

From reflex spasms.—It is very important to distinguish between spasms due to reflex irritation and those of cortical origin, which constitute the tics. Thus "tic douloureux," a reflex spasm arising from the irritability of the fifth nerve in cases of severe neuralgia, has nothing of the characters of the tics we are now considering, though, unfortunately, its name is apt to lead to confusion.

Reflex spasms of the sterno-mastoid, trapezius, and other muscles of the neck may all give rise to torticollis, and must be carefully distinguished from tics of cortical origin. A physiological as opposed to an anatomical, distribution of the movements will often be of some assistance. For instance, a coordinated action between different groups of muscles, such as the sterno-mastoid on the one side and the deep rotators of the neck on the other, is much more suggestive of cortical origin than of some local nerve trouble that has by coincidence happened to irritate two widely separated muscles which physiologically tend to act together.

Prognosis.—The outlook varies considerably for different cases. In children, simple tics frequently entirely disappear, but in adults the result is not so hopeful.

Those which have definitely come on during a period of special strain are more likely to disappear in favourable circumstances than those that have come on insidiously during the ordinary daily life. Those which are distinctly part of an hysteria may also do well. In many the movements diminish with improvement in the general health, but they show themselves again very readily during worry or excitement.

Treatment.—Any source of peripheral irritation which causes or aggravates the spasm must be removed; next, the general health must be attended to. Electricity is useful, but needs careful use. Where the movement tends to assume a tonic character, as in some of the cases of torticollis, faradism of the opposite muscles is sometimes beneficial, while galvanism may be applied to those at fault.

Carefully graduated exercises, whereby the patient learns to arrest some movements and to make others more regular, are often very useful, and may be practised in front of a looking-glass. Massage and general exercises are valuable in some cases, and in others, where neurasthenia or hysteria seems obviously to be at the bottom of the trouble, a course of Weir Mitchell treatment may be very beneficial.

—The treatment of tics by surgical measures is tempting, but must always be approached with caution, and the recollection of the necessity for this will often avert the disappointment of finding the spasm extending to other muscles, after the nerves to those originally affected have been cut. On the other hand, the spasms of reflex origin may often be much more favourable for surgical intervention.

NODDING SPASMS (SPASMUS NUTANS)

Rhythmical movements of the head, either of a nodding or side-to-side character, associated with a lateral or rotatory nystagmus, are frequently met with in infants. The origin is not clear, but the usual associated conditions are debility, teething, or rickets.

The prognosis is good, and in most of the cases the abnormal movements, both of head and eyes, cease as the child grows older. Cod-liver oil and other tonics are useful in treatment.

CHAPTER XLIII

MYOCLONUS; MYOTONIA CONGENITA; AMYOTONIA CONGENITA; FAMILY PERIODIC PARALYSIS

MYOCLONUS (PARAMYOCLONUS)

MYOCLONUS is characterised by rapid, sudden contractions of muscles resembling those obtained by an electric shock.

Etiology.—Nothing definite is known about its causation beyond the fact that debility and ill-health seem often to have preceded it. It sometimes occurs in several members of the same family, and it is apt to be associated with epilepsy.

Symptoms.—The characteristic symptoms are repeated, sudden, electric-shock-like contractions of muscles, chiefly of the limbs, less often of the trunk, and least often of the face.

The contractions are usually bilateral, and occur on both sides either at the same time or immediately after one another. If they are sufficiently strong and extensive the whole limb moves, or in severe cases the patient may be thrown down. The spasms usually cease during sleep, and are often ameliorated during voluntary movements. The reflexes are generally exaggerated, and the nutrition of the muscles remains good.

The seat of the disease is uncertain, but from its frequent association with epilepsy the cerebral cortex seems to be the most likely place.

Diagnosis.—The movements differ from those of *rheumatic chorea* in their suddenness, persistence, and distribution, while there are not the wasting and general symptoms met with in electrical chorea. The *tics* may be confused with myoclonus, but they are more purposive and more likely to be one-sided.

Prognosis.—There is no certainty about the course of these cases. In most of them the movements persist, but in a few they cease spontaneously or as the result of treatment.

Treatment.—In the absence of any discoverable source of irritation, the treatment must be conducted on general hygienic principles. If the spasms are very violent, sedatives, such as *hyoscyamus*, may be needed, and if *epilepsy* is present the bromides must be given.

MYOTONIA CONGENITA (THOMSEN'S DISEASE)

Etiology.—This disease appears to be due to some deficiency in development, and the symptoms generally appear during childhood. It is essentially a family disease, and is often handed on through several generations.

Pathology.—Some hypertrophy of the muscles often occurs, but there do not appear to be any changes in the central nervous system. It seems probable that this is one of the conditions which may be at any rate partially explained by a variation from the normal of the two main constituents of muscle fibre (p. 31), so that the power of the substance causing tonic contractions becomes the dominating feature.

Symptoms.—This disease is characterised by the occurrence of spasms at the beginning of a voluntary movement, which gradually pass off with the persistence of the movement.

On attempting to walk after resting, the legs are stiff and can at first only be moved with

difficulty, but after the movement is once established they become supple, and remain so until after another interval of rest.

The difficulty in starting a movement is well seen by asking the patient to close and open his eyes or his hands.

The muscles are usually well developed and hypertrophied, though there is often some degree of weakness.

Electrical reactions.—The reaction of the muscles is different from normal to both the faradic and constant currents. With the faradic current a longer, more tetanic contraction than usual is obtained. To the galvanic current the muscles are induly excitable, and the strength of the contraction obtained by the closure of the anode (ACC) approaches or becomes equal to that of the kathode (KCC). Direct percussion of the muscles also causes a slow contraction.

Prognosis and treatment.—The disease does not shorten life.

The only form of treatment which appears to produce any benefit is that of gymnastic exercises.

AMYOTONIA CONGENITA (MYATONIA CONGENITA)

In contradistinction to the condition known as myotonia congenita, described in the preceding section, there is an uncommon condition of obscure origin, which was first described by Oppenheim, and to which the title of amyotonia or myatonia congenita has been given.

Etiology. — The disease is met with in children, and in the majority of cases has undoubtedly existed from the time of birth. In a few the histories give rise to suspicions that the trouble has followed some illness, *e.g.*, measles in early childhood, but it is possible that the condition has pre-existed, and only become more obvious through the illness.

Symptoms. — The characteristic feature in these cases is absence of muscle tone, so that the limbs can be placed in all kinds of grotesque attitudes.

The distribution of the disability varies in different cases. In some the face, trunk, and limbs may all be affected, but in others the muscles



Fig. 92.—Extreme flexion of the ankle-joint in amyotonia congenita.
(From a case under the care of Dr. Essex Wynter.)

of the limbs, especially the legs, are the only parts to lose their tone.

The power of movement is greatly diminished, so that the child cannot stand or walk, and when the trunk muscles are toneless it may be unable even to sit up. If the face is affected there may be inability to close the eyes.

The muscles in the diseased areas are flabby, and a faradic current of unusual strength is neces-

sary to make them contract; moreover, currents of far greater intensity than can be borne by a normal child appear to cause no pain.

Diagnosis. — The principal diseases from which amyotonia has to be differentiated are



Fig. 93.—Grotesque position of the leg in the same case.

rickets, with which it may, of course, be associated, anterior poliomyelitis, and the myopathies.

From anterior poliomyelitis it is distinguished by its symmetrical distribution and by the absence of complete paralysis, for in amyotonia the movements are weakened but not lost. Further, there is not the wasting which occurs in acute anterior poliomyelitis.

From myopathies the distinguishing features

may be more difficult to discern, since the flabby muscles and generalised weakness are suggestive, but the loss of tone is such a prominent feature in amyotonia that it stands out above all else.

Prognosis.—The prognosis, as judged from the cases hitherto recorded, appears to be good. The muscles improve as the child grows, and they eventually become strong. In some instances contractures have been observed.

Treatment.—The main points in treating these cases are: (1) to look after the general nutrition; (2) to regulate the child's movements so that deformities and ill-shapen limbs are not allowed to develop from attempting to walk too soon; (3) to massage and move the limbs in order to prevent the formation of contractures and fixed positions.

FAMILY PERIODIC PARALYSIS

This disease, as the name implies, runs in families and is also transmitted from one generation to another. It occurs in both sexes.

Etiology.—The family and hereditary distribution is suggestive of some developmental disorder, while the periodic nature of the attacks seems to point to the likelihood of a temporary toxæmia.

Excessive fatigue, specific fevers, constipation, and indigestion are among the conditions that are thought to dispose to the onset of the attacks.

Symptoms.—The characteristic symptoms are temporary paralysis with loss of reflexes and electrical reactions.

The attacks occur from time to time after intervals of various lengths. The onset is generally during the night, the patient waking up to find that he cannot move his limbs. Observation has shown that power is generally lost in the legs first, and later in the arms and trunk. The muscles supplied by the cranial nerves are occa-

sionally weakened, in which case there may be symptoms of facial and bulbar paralysis.

The paralysis is of a flaccid character, and the deep reflexes and electrical reactions in the paralysed muscles are diminished or even lost.

The heart may be dilated and irregular in its action during the attacks.

Respiration is impaired by the paralysis extending to the trunk muscles, but it is kept up by the diaphragm, which, fortunately, appears seldom to be seriously affected.

Sensation and consciousness are not impaired.

After some hours, power begins to return in the limbs in the reverse order to that in which they were affected.

Prognosis.—The outlook, so far as life is concerned, is usually good, but occasionally death occurs during an attack.

Treatment.—On the assumption that some poison is present, the diet should be plain and the excretory organs should be kept active. Diuretics, especially citrate of potash, have been recommended for shortening and lightening the severity of the attacks.

No constant results seem to follow the administration of drugs, but, of course, strychnine, digitalis, and others are very necessary to assist a failing heart or an enfeebled respiration, and in the case of the latter it may be necessary to give oxygen and to perform artificial respiration as well.

CHAPTER XLIV

OCCUPATION NEUROSES

ALL occupations which necessitate for their performance a constant repetition of exactly the same movement, are liable, after a varying length of time, to become difficult or impossible to carry out. As a general rule, the more delicate and accurate the repeated movement requires to be, the more likely is the power of performing it to be lost, the more so if constant concentration and mental strain be required at the same time; and, further, individuals who are naturally of nervous temperament are the most likely to suffer.

Among those especially liable to be troubled in this way are writers, piano and violin players, and typists, but the affection is also met with in workers in many other trades, such as seamstresses, cigarette makers, dairymaids, and telegraphists. One of the most interesting that have recently come under the writer's notice is that of a man whose chief occupation was to sharpen razors, and who was unable to carry the blade smoothly backwards and forwards on the stone on account of sudden little spasms of the arm which quite spoilt the movement.

Writer's cramp, which is the most familiar form of occupation neurosis, occurs mostly in those who have to write a stiff, copy-book hand for many hours a day. It is distinctly connected with a prolonged cramped movement, for it does not seem to occur in those who write a free hand

from the shoulder. Clerks whose chief business it is to be continually copying long documents are among the most common sufferers.

Symptoms.—The symptoms are mainly those of cramp in the small muscles of the hand. After writing for a longer or shorter time, according to the severity of the case, the fingers become stiff and involuntarily separate themselves, so




Fig. 94.—Specimen of writing in writer's cramp.

that it is impossible to hold the pen, which is spasmodically dragged across the paper or even shot out of the hand; in some cases there is, every now and then, a strong flexor spasm.

Pain is a variable symptom, but there is often discomfort, with tingling and aching sensations. In some cases weakness is a more prominent feature than spasm.

At first the patient finds he cannot write so

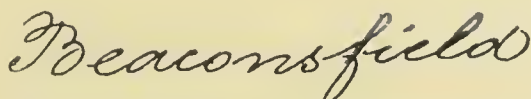


Fig. 95.—Specimen of former writing from the same case.

long or so quickly as he could formerly, and then the difficulty becomes greater and greater until at last his occupation is gone.

The accompanying figures show how the style of writing may change for the worse.

Most movements other than those concerned in writing can often be carried out with care, but occasionally some of the finer ones suffer a little, although usually not to anything like the same degree.

The electrical reactions are often unchanged.

In severe cases the muscles are over-excitabile; more rarely the reactions are diminished.

Seat of the lesion.—The cerebral cortex, the peripheral nerves, and the muscles themselves have all been regarded as at fault, but it is difficult to see how a change in a peripheral nerve can affect one set of movements to the exclusion of others. Nor is there any good evidence that the fault lies in the muscles.

The hypothesis most in harmony with the facts is that the trouble is of cortical origin and due presumably to exhaustion of the particular combination of nerve cells from which the movement originates. "The education of centres which may be widely separated from each other for the performance of any delicate movement is mainly accomplished by lessening the lines of resistance between them, so that the movement, which was at first produced by a considerable mental effort, is at last executed almost unconsciously. If, therefore, through prolonged excitation, this lessened resistance be carried too far, there is an increase and irregular discharge of nerve energy, which gives rise to spasm and disordered movement. According to this view, the muscular weakness is explained by an impairment of nutrition accompanying that of function, and the diminished faradic excitability by the nutritional disturbance descending the motor nerves."—Gay (quoted by Osler).

In others the loss of movement is not so strictly limited, and pain along the course of a nerve, with perhaps some slight evidences of a muscular wasting and changes in electrical reaction, seems to point to the possibility that the trouble is connected with the peripheral nerves.

Diagnosis.—When a patient complains of difficulty in writing, it is necessary to exclude other possible causes before concluding it is a case of writer's cramp.

1. Examine the hand and wrist for any local trouble, such as teno-synovitis.

2. Ascertain if there are any signs of localised neuritis, as shown by weakness in all movements, pain and wasting, or of multiple neuritis with a tendency towards a symmetrical distribution.

3. Remember that tremor from any cause will spoil the writing, and bear in mind, among other things, the possibility of commencing paralysis agitans, disseminated sclerosis, and general paralysis of the insane.

Prognosis.—The prognosis is not good, so far as the possibility of recovering power to perform the particular movement is concerned. It is very seldom indeed that anyone who develops a definite occupation neurosis is ever able to earn his living again by that particular movement. Cases in which the lesion seems to be in the periphery are probably more favourable than those of cortical origin.

Treatment.—Complete rest from the particular occupation at the very first sign of the trouble gives the best chance of ultimate recovery. Occupations involving other and coarser movements may be continued, provided that care is taken to avoid tiring the affected limb. Locally, massage, hydropathic treatment, and galvanism are all beneficial. The faradic current should generally be avoided.

If the pain and spasm persist when the occupation has been left off, such drugs as bromides, phenacetin, and antipyrin may be given, but the main effort in general treatment should usually be towards improving the health by rest and a nutritious diet with plenty of milk and, if needful, cod-liver oil. Various devices are generally adopted by the sufferers to put off the evil day of relinquishing their work. In writer's cramp the patient finds he can get along better for a time by holding the pen between the first and second

fingers, and from this he takes to a very thick penholder, which he grasps with the whole hand.

Unfortunately, any impairment in the quality and quantity of work causes a failure to attain the standard that is demanded, and no device can more than temporarily hide this deterioration.

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CHAPTER XLV

NEURASTHENIA

NEURASTHENIA is the term applied to a combination of symptoms which appear to depend upon a modification of the nutrition of the nerve elements.

It is a "functional" disorder in connection with which no organic changes can be recognised, and, although its borderland is ill-defined, there is nevertheless a large group of cases which are conveniently classed under this heading.

Etiology.—Both sexes suffer from neurasthenia, though, on the whole, more women are affected than men. Overwork, both physical and mental, appears to be one of the most frequent causes, but any debilitated condition such as follows a severe illness may be the starting-point from which the symptoms arise.

Persons with neurotic dispositions, whether inherited or acquired, are very liable to become neurasthenics.

Quite some of the worst cases follow injury, and are known under the term traumatic neurasthenia.

It seems probable that an auto-infection from the alimentary canal or from deranged chemical processes in other parts of the system is frequently a contributory agent in the causation, though it is difficult to be sure of the relations between cause and effect. Symptoms of gastric and intestinal disorder are frequently present, and often

add largely to the mental discomfort of the patient. At present it is not wise to be dogmatic, but it is better rather to look upon neurasthenia as being due to deficient nutrition of the nervous system, and, in trying to get rid of the disorder, to bear in mind the many possible ways in which such deficient nutrition may be brought about.

Symptoms.—The symptoms may be divided into *mental* and *physical*.

Mental symptoms.—Impulses reaching the brain of the neurasthenic cease to maintain the relative proportion which they have previously borne to one another. Incidents of little importance are magnified, especially those connected with health. The slightest ache or pain is often the starting-point of some imaginary dreadful disease; an unimportant lapse of memory is thought to signify commencing cerebral disorder; while in the mental background there is frequently an indescribable fear of impending trouble which entirely destroys all peace of mind. These symptoms may be shortly summed up by the statement that the patient worries unnecessarily and loses confidence in himself.

Nervousness in the presence of people, fear of crossing open places, and a dread of going mad are common symptoms. There is difficulty in concentrating the mind upon any intellectual work, which often erroneously leads to the idea that the memory is failing, and there is a fear of making mistakes in work, though, as a matter of fact, such mistakes are not generally made.

Throughout it is a fear of what he may do and of what may happen, rather than of what he does and what does happen.

Such a state of mind may become worse and pass into the domains of hypochondriasis and melancholia. It will also be seen that hysteria and neurasthenia have no very definite dividing line.

Physical symptoms.—On the bodily side the symptoms are most various.

Pains, crawling sensations, and other peculiarities are complained of both in the head and body. The headache is often characterised by a sense of pressure at the vertex, but at other times it is indistinguishable from the frontal, vertical, and occipital headaches due to other causes. Tenderness and "weakness" of the spine are frequently complained of. In the abdomen the common accompaniments are indigestion, with which is often associated some dilatation of the stomach, and constipation. Flatulence is often a most troublesome symptom, especially in women. Added to these there are "sinking" and "fainting" feelings. The heart generally beats rather faster than normal, and the pulse-rate is easily varied by nervousness or excitement.

The limbs soon tire, and after walking there may be a dull pain in the lumbar region.

The patient shows great anxiety to detail all his troubles, and generally makes a long list of them so that he may be certain to omit nothing.

Attempts have been made to divide up cases of neurasthenia, according to the seat of the most prominent symptoms, into cerebral, spinal, gastric, and cardiac varieties, but such a division is too artificial to be of practical use.

Physical signs.—On examination, no signs of organic disease will be discovered. It is usual to find a moderate increase of the deep reflexes, with the plantar reaction of flexor type, or in some cases no reaction at all may be observed. There is no ankle-clonus, though a "pseudo" clonus, in which there are two or three feebly-sustained clonic contractions that cease when the foot is fully flexed, may be obtained.

The limbs are feeble generally, but there is no definite paralysis.

Examination of the sensory system does not reveal much except where cases are bordering on hysteria, and then some of the different forms of anæsthesia peculiar to the latter may be found.

As regards the eyes, the pupils are generally large, but they are equal and contract to light and during accommodation.

The movements of the eyeball are normal, and there are no changes to be observed in the fundus. There is, however, often an error of refraction, to which allusion will again be made when speaking about treatment.

The history reveals no symptoms that can be definitely associated with organic changes.

There will have been no double vision, no loss of control over the sphincters, and no fits.

Diagnosis.—It will be seen from what has been written above that symptoms indistinguishable from those of neurasthenia are in a general way apt to be present at the beginning of many diseases, some of the principal of which it is as well to bear in mind while examining the patient.

General paralysis of the insane may begin with symptoms which exactly resemble those of neurasthenia, and until some more definite signs develop it may be impossible to be sure of the diagnosis. As a rule, however, the resemblance is more apparent than real. The important point to which attention should be particularly given is the exact nature of the mental change that is present, especially as regards character and work. The general paralytic is not only unable to work so well, but he loses his skill and also his finer sentiments. The neurasthenic is over-anxious about himself, worries, and is unable to keep his attention fixed for long, but he does not show the tendency to mental degeneracy that is seen in the general paralytic. He may be over-anxious about his work and afraid of making mistakes, but, as a matter of fact, he seldom does make

them; while, on the other hand, the failing mental powers of the general paralytic are often obvious to everyone else but himself. The presence of any signs of organic disease, such as inequality of the pupils, with loss of reaction to light, of course at once excludes the existence of neurasthenia alone.

Neurasthenics are often depressed, and if that depression is accompanied by suicidal tendency and delusions, then the case should no longer be regarded as one of ordinary neurasthenia, but should rather be included under "*melancholia*," however slight, so that proper nursing and supervision may be provided to ensure the best chances of recovery. The presence of definite anæsthesias and paralyses will help in the differentiation of *hysteria*, but often no sharp definition can be drawn. When the patient complains of being tired after walking a little way it is well to bear in mind the possibility of *myasthenia gravis*, which in its less pronounced forms may be confused with neurasthenia. The importance of differentiating *disseminated sclerosis* from functional disease is insisted on elsewhere.

Treatment.—The objects to be kept in view in the treatment of neurasthenia are:

1. To raise the nutrition of the body generally and of the nervous system in particular.
2. To remove any deleterious substances which may be acting as poisons.
3. To remove any irritation from the periphery.

The methods to be adopted to improve the general nutrition will vary with the severity of the case. When the symptoms are slight, of recent onset, and accounted for, perhaps, by some temporary excessive mental strain, an ordinary holiday, with plenty of food and a simple tonic containing *nux vomica* or iron, may suffice. Where the symptoms are severe and of longer standing,

melancholia

Hysteria

Myasthenia Gravis

Disseminated Sclerosis

a "rest cure" on the lines laid down by Weir Mitchell is by far the most successful method of treatment.

The principal factors in the Weir Mitchell treatment and its modifications are isolation, diet, massage, regulation of the bowels, and the confidence of the patient in his medical attendant.

Isolation, in any but the mildest cases, should be complete, at any rate for a time. The patient must be removed from home surroundings, must see no friends, and must receive and write no letters. The nurse should be one who is accustomed to dealing with these cases.

This isolation is very soothing to the patient, and enables the nervous system to obtain complete rest. Where the isolation cannot be obtained in a special room, good results may be got from screening off the bed from the rest of a ward.

The main article of diet should be milk, which can, by gradually increasing the amount, be taken in quantities varying from three to six pints daily. The rest of the diet, which should be liberal, must be arranged according to the requirements of the individual and the quantity of milk which is being taken, special modifications being required if the patient is obese. Indigestion, which is present in so many cases before treatment, is often due to fatigue, and disappears as soon as the régime is fairly started.

Under a diet of this kind the patient increases in weight, and the nervous system shares in the extra nutrition which is thus obtained.

The massage should be carried out regularly, but its daily duration and intensity must be regulated for each patient.

The massage takes the place of exercise. By it the circulation of the lymph and blood is increased, and its effects are such as to enable the patient to take and digest greater quantities of food than would otherwise be possible.

Regulation of the bowels is important. The symptoms of neurasthenia are in a considerable proportion of cases associated with constipation. Indeed, some writers consider that neurasthenia is nearly always the result of a toxæmia which arises within the intestines, and which, presumably, is made more intense by constipation. In any case, many of the patients are greatly worried by this condition of things, which has "got on their nerves," and they have usually tried to correct it in a spasmodic manner, which only brings temporary relief.

The choice of purgatives is naturally very large, but must be adapted to the object in view, which is that of restoring regular rhythmical actions. As a general rule the author finds that saline draughts, to which may be added a little nux vomica, taken twice or three times a day about half an hour before meals, and directly followed by a glass of hot water, are efficacious. Until regularity is restored, enemata given frequently are often very useful.

Errors of refraction, if present, should be corrected, since a continuous strain of the ocular muscles often leads to irritability and exhaustion.

The confidence of the patient in his medical adviser is all-important, since it conduces to a tranquil mind and an unquestioning obedience, without thought or argument, to the various details of the treatment.

CHAPTER XLVI

HYSTERIA

UNDER the term hysteria are included a variety of well-recognised symptoms, which appear to depend upon abnormalities of the irritability and conductivity of the nervous tissues, so that the effects produced by various impulses are greatly modified, and different functions which normally act together are liable to become dissociated. The processes connected with memory associations, which underlie the "will," are also often affected, so that volition is either unnaturally weak or absent.

These modifications in the irritability of the nervous tissues are not associated with any changes in structure that can be detected by the known methods of investigation, and, indeed, it seems probable that they are produced by chemical processes which are never likely to be detected by the microscope. It is obvious that symptoms depending on such causes cannot have any sharply defined boundaries, for many organic diseases are naturally often preceded by similar changes in the irritability and conductivity of the nervous system before any signs of organic destruction can be detected, as also are many of the more severe "functional diseases," among which are included some of the forms of insanity.

Etiology.—Hysteria occurs more frequently in females than in males, although the latter are by no means exempt; quite young children may also be subjects of the disease. It is common for

the symptoms to appear about the time of puberty or soon afterwards. Heredity is an important factor, as also are the natural temperaments of different nationalities. The first symptoms may set



Fig. 96.—Hysterical contractures in a child.
(From a case under the care of Mr. T. H. Kellogg.)

in after a mental or physical shock; and, lastly, it must never be forgotten that some definite organic lesion may underlie hysterical manifestations.

Symptoms.—The symptoms are so numerous and ill-defined that it is very difficult to classify

"pseudo" ankle-clonus, which is characterised by a few short taps, generally ceasing when the foot is fully flexed. This pseudo-clonus, as it is called, is not of much value, since an incomplete clonus is often obtained in organic disease. On the other hand, a strong and sustained clonus is greatly in favour of organic disease, though not necessarily conclusive.

Plantar reflex.—The reaction of the plantar reflex is very important, for an undoubted extensor response is a conclusive indication of organic disease.

In many instances of functional disorder there are times when no plantar reflex of any kind can be obtained, and although such cases are suggestive of functional disorder, too much stress must never be laid on a negative result, since experience shows that a plantar reflex which is either flexor or is not obtainable one day may on another give a definite extensor reaction.

One may sum up the comparative importance of the different reactions of the plantar reflex as far as the diagnosis of *hysterical* paralysis is concerned as follows:—

EXTENSOR RESPONSE.—Indicates conclusively the presence of organic disease of the motor tracts.

FLEXOR RESPONSE. — Suggestive of the absence of organic disease of the motor tracts.

ABSENCE OF RESPONSE.—Suggestive of functional disease, but care must be taken to see that the absence is not merely accidental or temporary.

The sphincters.—There may be retention of urine with overflow-incontinence, but never the true incontinence that occurs when the lumbar enlargement of the cord is damaged.

The larynx.—Paralysis in the laryngeal muscles is common, and gives rise to aphonia, which often

disappears as suddenly as it appears. It is always the adductor muscles that are affected, so that the cords are widely separated and cannot voluntarily be brought together, although they move freely during respiration and coughing, both of which acts can be performed automatically, without the intervention of the will.

Paralyses of the larynx and, more rarely, those of the face are the only ones likely to occur in the distribution of the cranial nerves. The squints and ptosis which occur are usually due to spasms, and are referred to in the section below.

GENERAL SUMMARY OF THE DIAGNOSIS OF HYSTERICAL PARALYSES

The procedure in diagnosing hysterical paralysis does not differ from that required in the diagnosis of loss of power from other causes.

The possibilities must all be carefully considered, and the diagnosis of hysteria only arrived at after rigid exclusion of everything else. Assuming the patient to have *lost power in the legs*, the following questions must be asked:

1. Is the lesion situated in the upper or lower neuron? (Upper, on account of increased or normal tendon-reflexes, absence of wasting, and electrical changes in the muscles, with possibly some active contractures.)

2. Is the lesion in the spinal cord? Consider the reactions of the reflexes (knee-jerks, ankle-clonus, and plantar). If the lesion is due to an anatomical change in the lateral columns, these should all behave consistently. An organic lesion of the lateral columns which produces exaggeration of the knee-jerks should, in the ordinary course, also give rise to an extensor response of the plantar reflex, and in all probability an ankle-clonus. If, therefore, the signs are contradictory, as they are when an exaggerated knee-jerk exists with a flexor or an absent plantar response,

suspicion may be aroused as to the likelihood of an anatomical lesion being present.

On the other hand, the absence of knee-jerks entirely negatives hysteria, as also does a typical extensor response of the plantar reflex.

3. *What is the condition of the sphincters?* Retention of urine is not conclusive evidence of either functional or organic disease, but true incontinence is indicative of organic change.

4. *If anæsthesia is present, what is its distribution?* The anæsthesia of organic disease will be distributed according to anatomical rules. In hysteria the intensity and distribution of the anæsthesia may vary from time to time, and frequently does not correspond in any way to the distribution of any organic lesion.

Additional considerations.—Gait.—The gait in hysterical paralysis assumes a variety of forms, some of which are obviously too complicated to depend upon organic lesions. In hysterical hemiplegia the affected leg is dragged along with the upper surface of the toes on the ground, instead of being stiffly swung round from the hip-joint as it is in the organic spastic cases. The hysterical paraplegics generally shuffle along without being able to raise the soles of the feet from the ground.

Rigidity.—The results of rigidity of hysterical paralysis frequently do not correspond to those of organic disease, where the position of the limbs is governed by anatomical rules. Thus in hysterical hemiplegia, the arm may be extended or otherwise fixed in a manner quite different from that so well known in organic cases.

OVER-ACTION

Tremors.—The intensity of hysterical tremors may vary from the finest to the coarsest movements, and, like other hysterical manifestations, they are apt to differ very widely from time to time. They often closely simulate the tremors of

organic disease, but can generally be distinguished from them by the absence of other symptoms which would naturally be associated with the tremor, and by the failure of the tremor to behave continuously like those known to be associated with organic disease.



Fig. 97.—Hysteric spasm of the tongue. (*Pierre Marie*.)

Spasms.—Localised spasm of muscles is of common occurrence, and may be observed in connection with muscles supplied by both cranial and spinal nerves.

Of the muscles supplied by cranial nerves, the most commonly affected are those of the eye, and more rarely those of the tongue (Fig. 97) and the jaw.

The eyes.—Spasm of the muscles of accommodation is frequent, as also are spasmodic ptosis and convergent strabismus. The ptosis, which at first sight may appear to be a paralytic manifestation,

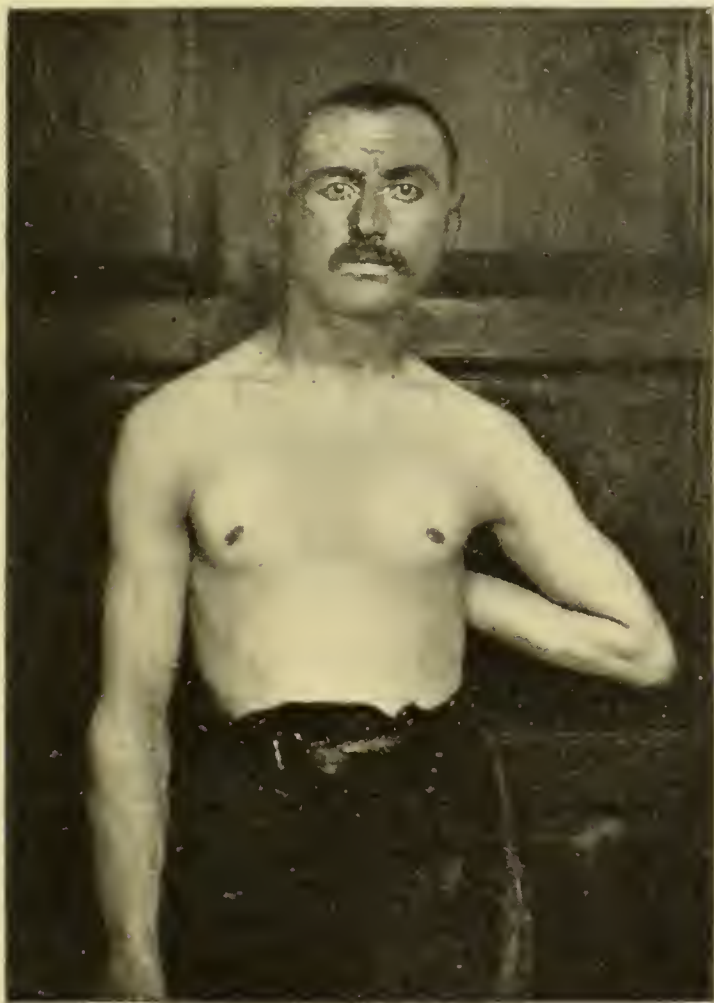


Fig. 98.—Hysterical spasm of the arm. (*Pierre Marie.*)

is due to spasm of the orbicularis palpebrarum, and is unaccompanied by any compensatory contraction of the occipito-frontalis, such as occurs with the paralytic form of ptosis. In a few rare instances, hysterical ptosis due to relaxation of the levator palpebræ has been recorded.

Hysterical *contractures* of the *limbs* (Fig. 98) are often exceedingly tense, and any endeavour to relax them by force only increases the opposition, nor does an attempt to approximate the points of insertion of the contracted muscles aid their relaxation, as it does in the cases of organic origin. Relaxation is, however, generally effected during sleep, and by the aid of an anæsthetic.

Contractures are especially common in the feet, knees, and hips, and may be accompanied by great pain.

Catalepsy, during which the limbs remain for a long time in any position in which they are placed, sometimes occurs.

Spasms of respiratory muscles.—Irregular spasmodic action of the muscles of respiration is associated with hysterical hiccough, yawning, cough, and globus. Hysterical dyspnœa may also be present.

Spasm of the œsophagus with difficulty in swallowing may suggest the presence of a stricture of organic origin.

Convulsions.—Hysterical convulsions, which in many respects bear a close resemblance to epileptic fits, may occur. They are usually ushered in by some hysterical sensation, as the globus hystericus, flatulence, or palpitation. Generalised tonic spasms then occur, and the patient falls or slides to the ground, but seldom with the sudden helplessness that is so characteristic of epilepsy. The tonic stage is succeeded by clonic movements which are often very wild and uncontrolled, and in severe cases opisthotonos and different elaborate postures of the body are assumed. After a variable time, usually much longer than in epilepsy, the movements cease, and the normal mental condition gradually returns. The main diagnostic points between these fits and epilepsy are mentioned in the chapter on Epilepsy (p. 373).

Lastly, among the infinite variety of spasms

and movements that are met with may be mentioned the dancing movements known as saltatory chorea.

SENSORY SYMPTOMS

Anæsthesia.—Anæsthesia is the symptom most constantly present in hysteria. The variability of its distribution and intensity is well known, but the most frequent distributions are over one half of the body, the distal portion of a limb, or a segment of a limb. It may also be present in irregular patches.

In hemianæsthesia the loss may be complete for all kinds of sensation, or it may be partial, or there may be loss for one kind of sensation and not for another. The sensory loss usually extends to the middle line of the body and affects mucous membranes as well as skin.

The special senses are also often defective at the same time. The anæsthesia may disappear during sleep. Sometimes a touch on one limb is felt on the opposite limb (allocheiria).

Hyperæsthesia.—In some instances sensation is abnormally acute over circumscribed areas, and pain and tenderness may be prominent symptoms. Pains may simulate those of rheumatism, migraine, and angina; headaches are common, and may be severe enough to suggest the presence of a tumour. Tender points (hystero-genetic spots), pressure on which may start a convulsion, often exist over various vertebræ, below the mammæ, and over the ovarian regions.

Visual symptoms.—Allusion has already been made to the spasms of the ocular muscles. The fields of vision are usually contracted in a manner characteristic of the disease: there is constriction of both fields, but more so of one than the other, the constriction being greatest on the side of the anæsthesia. This condition is known as crossed amblyopia. The chart of the field of vision in these cases often has a spiral or corkscrew appear-

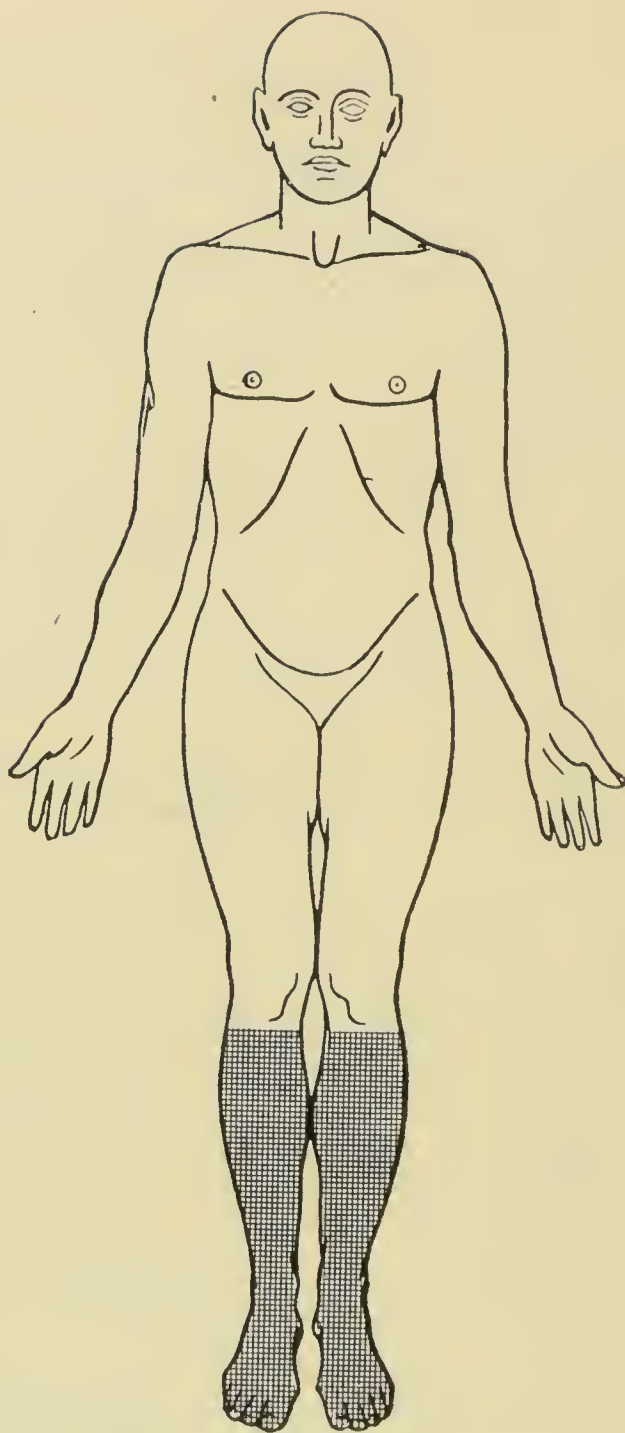


Fig. 99.—A type of hysterical anaesthesia.

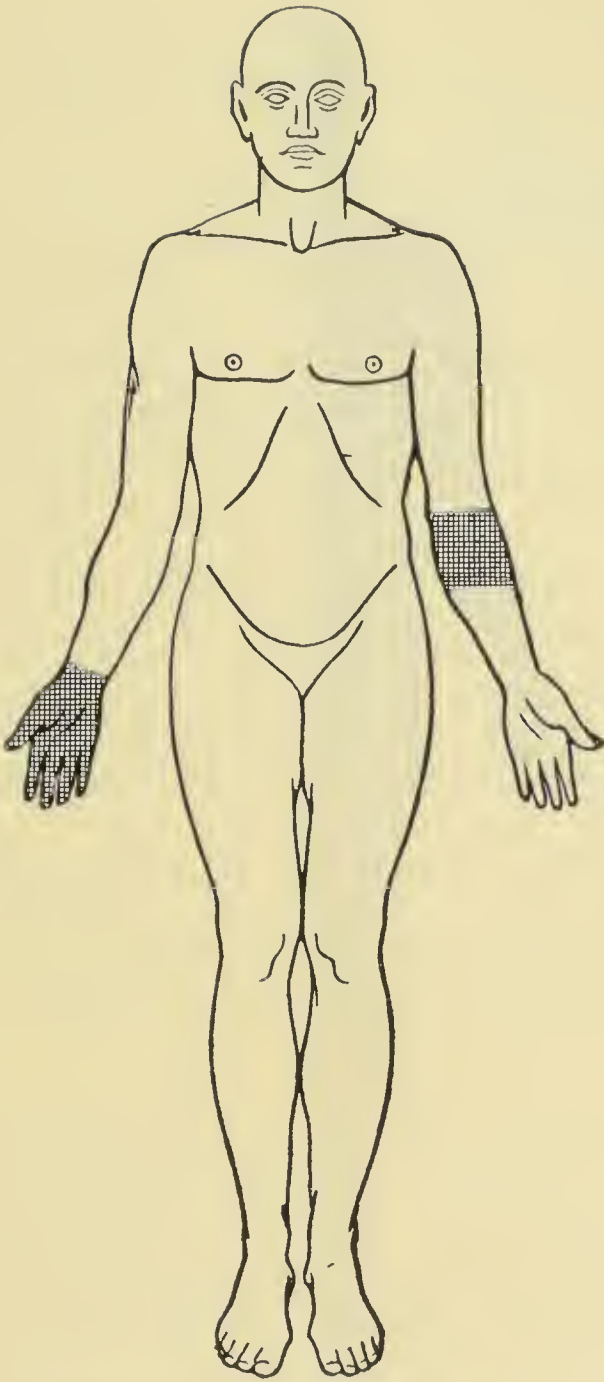


Fig. 100.—A type of hysterial anæsthesia.

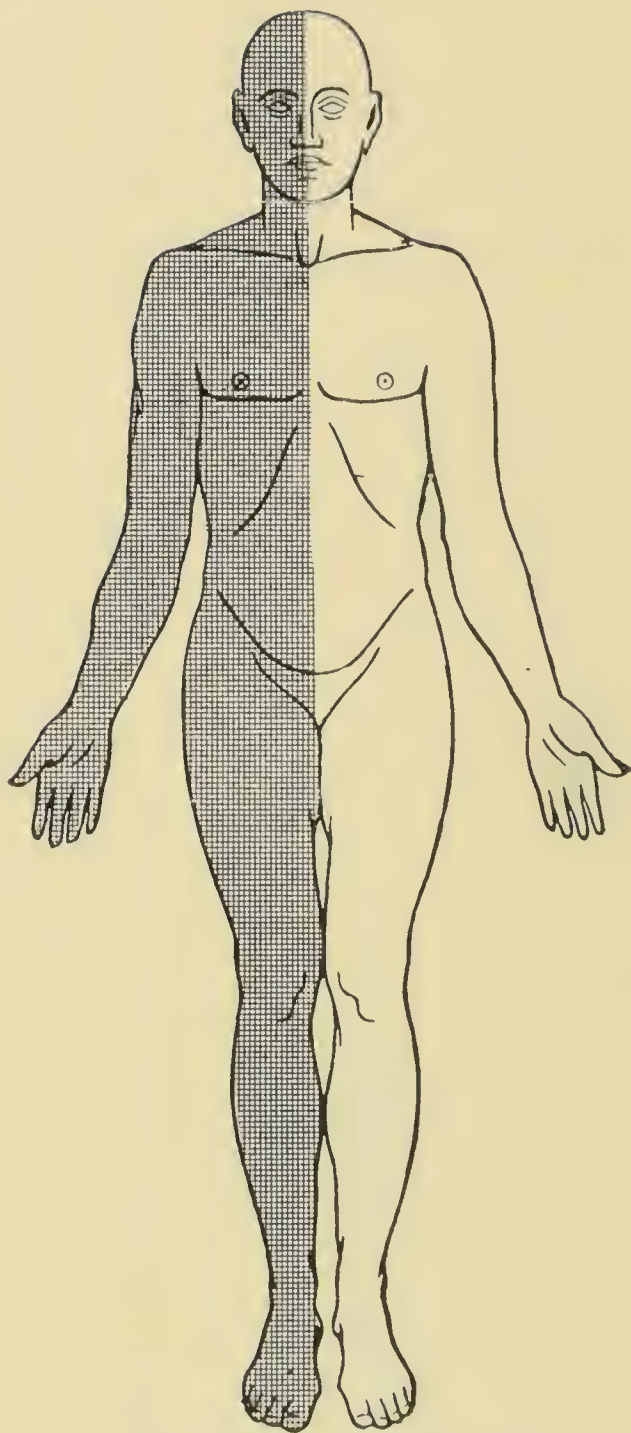


Fig. 101.—Hysterical hemianæsthesia.

ance, owing to the field becoming progressively smaller as it is being taken.

Hemianopia from hysteria, if it occurs at all, is so rare that it need not be considered. Recognition of colours is sometimes modified so that everything appears a uniform grey (achromatopsia). Short of this the field for red is the last to be lost, whereas in organic disease it usually fails first. Complete hysterical blindness is rare.

Deafness and loss of taste may also be present on the same side as the hemianæsthesia. When loss of smell occurs it is generally bilateral.

Gastric symptoms.—There may be loss of sensation of hunger, or there may be undue craving for various foods. Sometimes there is vomiting. Obstinate constipation may be present.

Vasomotor disorders, such as flushing, erythematous eruptions, and even cutaneous hæmorrhages, may occur. Dermographism, the condition in which wheals are readily brought out by drawing the finger-nail across the skin, as in the case of those suffering from urticaria, may also be present.

Temperature.—The temperatures of extraordinary height that are occasionally recorded in hysteria are probably of artificial production. A rise of temperature over any length of time should arouse suspicion of the existence of underlying disease.

MENTAL SYMPTOMS

Capriciousness, rapid change of moods, unreasonable likes and dislikes, are among the most prominent mental symptoms of hysteria. There is a general exaltation of the emotional side of the mind, with weakening of the power of judgment. In short, the nervous system is unstable, and the most various reactions may result. Craving for sympathy sometimes exists to such a degree that self-mutilation is practised in order to gain it. In the more severe cases, cataleptic conditions

may be met with. "Double personalities" also occasionally occur.

While there is, of course, no sharp line between hysterical symptoms and those of insanity, there is sometimes a tendency to minimise those of the latter by cloaking them under the term hysteria, often to the ultimate detriment of the patient.

Diagnosis.—Hysteria may simulate the effects of almost any organic disease, and the diagnosis in some cases may be very difficult, for beyond the decision that this or that symptom is "functional," one has to consider whether there is a background of organic disease upon which the hysterical symptoms are superimposed. The only way of avoiding mistakes is to search thoroughly for evidence of organic disease in the past history and present condition of the patient. The chief signs of organic disease which should be looked for are paresis of ocular muscles, fixed pupils, nystagmus, hemianopia, optic neuritis and atrophy, loss of tendon reflexes, extensor response of the plantar reflex, a true ankle-clonus, and incontinence of urine. It may be said that evidence can be adduced to show that all of these signs can appear in hysteria, but the occurrence of, at any rate, most of them is exceedingly rare, and for practical purposes it is safe, whenever the nature of the case is doubtful, to regard them as indications of organic disease.

Stress has elsewhere been laid upon the danger of confusing disseminated sclerosis with hysteria, and the diagnosis of the hysterical convulsions from epilepsy has also been discussed.

Prognosis.—The prognosis is, on the whole, good. Many cases recover spontaneously, or respond rapidly to suitable treatment, but others may take months or even years before they are cured. All the symptoms may disappear quite suddenly.

Treatment.—Mild cases of hysteria recover

with improvement in general health and change of surroundings, especially if the latter is accompanied by effective moral control. In others a course of electrical treatment hastens recovery. Where the symptoms are persistent and at all severe, a course of Weir Mitchell treatment is necessary, for the details of which the reader is referred to the chapter on Neurasthenia (p. 416).

In entering upon the treatment of any case of hysteria it is necessary to remember that the patients are very susceptible to suggestion, and at the outset the plain facts of the case and the objects of the treatment should be explained to them. Psychotherapy is, indeed, one of the most important factors in treatment, but for success it is first necessary that the confidence of the patient should be gained.

Hypnotism may occasionally be useful in carefully selected cases, but it has not found general favour in the treatment of this disease, and should only be used in exceptional circumstances.

Valerian and asafoetida are both useful drugs, as also are general tonics, such as iron and arsenic, when there is anæmia or some other indication for their use.

Hydrotherapeutics may be a valuable addition to other forms of treatment, and the faradic current is very helpful in aiding to cure local paralysis and anæsthesia.

CHAPTER XLVII

NEURALGIA; HEADACHE; MIGRAINE

NEURALGIA

Etiology.—Neuralgia is the term given to recurrent nerve pains which do not depend upon any obvious organic lesion, but it is often used in a more general sense to indicate many other kinds of nerve pains.

The cases may on this basis be divided up into *primary* and *secondary*.—

Primary neuralgia, with which is associated no discernible organic lesion, is especially associated with exposure to cold, debility, toxæmias, arterio-sclerosis, eye-strain, and irritation of the peripheral ends of the nerves, *e.g.* in the case of carious teeth.

Secondary neuralgia often accompanies inflammation, injuries, and degenerations of nerves. Of the last, the pains of tabes and those accompanying and following herpes zoster are good examples.

Symptoms.—Pain is the most prominent symptom; it is generally intermittent, of a sharp, stabbing nature, and sometimes tends to recur with great regularity at certain times in the day or night. The skin over the painful area is often tender to the touch, and in severe cases the nutrition of the hairs suffers, so that they turn white or fall out. Crops of herpes may appear over the painful area.

It is very common to find "painful spots"

where the nerve twigs are superficial and can easily be pressed upon.

Attacks of very acute pain may be accompanied by a reflex spasm of the muscles situated near the painful part. This is especially notable in severe cases of trigeminal neuralgia, and constitutes the condition known as tic-douloureux.

Diagnosis.—Whenever possible, it is necessary to distinguish a primary neuralgia from one of secondary origin. Endeavour must therefore be made to exclude the probability of inflammation, pressure, injury, or degeneration as causes.

A line cannot always be drawn sharply between neuralgia and neuritis, but in well-defined cases the intermittence of the pain, the absence of anæsthesia, of muscular wasting, and of any changes in the electrical reactions of the muscles suggest neuralgia.

The lightning pains that occur in the course of tabes are apt to be diagnosed as simple neuralgia before other signs become manifest. Gummata and other tumours must also be considered as possible sources of irritation.

Prognosis.—Usually, slight attacks of neuralgia, such as are commonly complained of by people who are feeling “run down,” disappear when the general health improves.

Severe and persistent pain is often far more difficult to cure, especially when it sets in in the course of the trigeminal nerve, and in many cases of the latter no other treatment short of removing the Gasserian ganglion may suffice.

No hard and fast rules can be given, for the likelihood of recovery in every case can only be gauged after the effects of treatment have been ascertained.

Treatment.—The objects aimed at are—(1) improvement of general health, (2) removal of poisons such as those of gout or lead, (3) removal of sources of irritation of the nerve-endings, and

(4) administration of such drugs as are known to influence the pain.

The methods of attaining the two first objects depend on the practice of general principles which need not here be discussed in detail; they include, chiefly, attention to the digestion and the general hygiene of the patient and the correction of anæmia, if present. The removal of all sources of peripheral irritation is specially important in neuralgia of the trigeminal nerve, which is often the result of eye-strain or decayed teeth; these will again be alluded to in the section on trigeminal neuralgia.

The drugs from which relief from neuralgia can often be obtained are numerous, but are not very dependable.

Phenacetin, antifebrin, and antipyrin, with preparations of a kindred nature, are among the most popular.

Iodides are useful in certain cases where arterio-sclerosis and chronic rheumatic processes appear to be the cause of the pain.

Gelsemium sometimes cures the pain of facial neuralgia most successfully, but it is generally necessary to give it in doses of sufficient strength to produce unpleasant physiological effects.

Morphia will, of course, relieve the pain, but it should, needless to say, be reserved for the most occasional use only.

Alkalies in large doses, especially ammonium carbonate and ammonium chloride, are often useful.

Externally, ointments of menthol and other substances that dull the sensibility of the nerve-endings are valuable; and where there are any chronic inflammatory changes round the nerve, massage and galvanism may afford relief.

TRIGEMINAL NEURALGIA

Neuralgia of one or more branches of the fifth nerve is a very common complaint. The pain is

generally at first confined to one branch, and the supra-orbital is the one which is most commonly affected. In its slighter forms it responds readily to treatment, but in its more severe forms it is most difficult to cure.

Etiology.—Of the peripheral forms of irritation, those most likely to induce it are eye-strain from errors of refraction, decayed teeth, and diseases of the nose, but it frequently happens that no source of the trouble can be traced.

Symptoms.—The pain, which recurs in paroxysms, is often started by a movement of the jaw, as in talking or eating, and reflex spasm of the facial muscles is an occasional accompaniment, and constitutes the well-known condition of tic-douloureux. When the paroxysms are very severe the pain may extend to the neck and arm and even over the trunk. There is often hyperæsthesia over the area of pain, and specially tender spots where the nerves emerge from foramina. The hairs of the eyebrows may turn grey or fall out, as also may patches of hair on the head.

Diagnosis.—The possible existence of a source of peripheral or central irritation is the first thing requiring investigation. The teeth should be carefully examined, but even when they are decayed the greatest care must be taken before concluding that they are the source of the trouble, for in many cases a large number of teeth have been sacrificed to no purpose.

The effects of application of heat or cold to the individual teeth is a test largely relied upon by dental surgeons. As already mentioned, it often happens that no source of nerve irritation can be found, and recourse has then to be had to empirical treatment by some of the drugs mentioned in the last section.

If no relief can then be obtained, the question of an operation will arise. Where the pain is limited to the course of a single branch, excision

of a piece of the nerve is a simple operation which may be followed by relief, at any rate for a time; but when the pain is distributed over the area of two or all three of the roots, a more radical operation such as removal of the Gasserian ganglion is the only thing likely to give relief.

NEURALGIA OF THE BRACHIAL PLEXUS

Neuralgia in the course of the brachial plexus is not very commonly met with. The general signs of neuralgia elsewhere are applicable here, but particular care must be taken before making the diagnosis to exclude the presence of organic disease of the cord or meninges, of tumours in the thorax, and of toxæmias of some constitutional condition, such as renal disease or diabetes.

INTERCOSTAL NEURALGIA

Neuralgia of the intercostal nerves is frequently met with. The pain which follows the course of the intercostal nerves is apt to be more continuous than in many other varieties, but is subject to acute exacerbations.

Tender points are generally present (1) close to the vertebral column where the posterior branches come to the surface, (2) in the line of the axilla corresponding to the area supplied by the lateral branches, and (3) where the anterior cutaneous nerves pierce the muscles to reach the surface of the chest.

Intercostal neuralgia often precedes the appearance of herpes, but in these cases it is scarcely correct to speak of the condition as neuralgia, since it has been shown that the herpes is accompanied by hæmorrhagic and inflammatory changes in the posterior root ganglion. The intercostal pains may persist for a long time after the herpetic eruption has subsided.

In making a **diagnosis** the principal conditions from which intercostal neuralgia must be distinguished are (1) pains from irritation or de-

generation of nerve roots, as may occur in tabes or tumours of the cord, (2) referred pains from visceral disease, (3) myalgia, in which the pain originates in the muscles.

HEADACHE

To ensure any success in diagnosing and treating headaches it is necessary to take a wide view of their causation, and not to attribute them all to some one special fault, such as an error of refraction or an unsuitable diet.

It is when headache is the main or only complaint that difficulty is most likely to arise, for when it is accompanied by other definite symptoms it is not generally difficult to assign to it its proper place in the disease.

The chief conditions in which headache is likely to appear as the most prominent symptom may be classified as follows:

1. Intracranial disease.
2. Disorders of the urinary system.
3. Disorders of the cardio-vascular system.
4. Disorders of the blood.
5. Migraine and toxæmias.
6. Eye-strain.
7. Reflex causes.

Routine examination of all cases is necessary if mistakes are to be avoided, for neither the position nor the character of the headache can be relied on as a guide to the cause, nor can a headache which is paroxysmal be safely ascribed offhand to an evanescent cause.

At the onset it is necessary to make sure that the pain is of the nature of "headache" and not of neuralgia, since the causation and treatment of the latter are often very different.

So far as the nervous system is concerned, unless the history specially points in that direction, it will generally be sufficient in the first place to examine the optic discs, the reaction of

the pupils, the ocular movements, and the deep reflexes—bearing in mind the possibility of the existence of intracranial growths, cerebral syphilis, tabes, and general paralysis of the insane—and to follow up the investigation in greater detail if any suspicious signs are elicited. The results of the examination of the urine will indicate the presence or absence of disease of the kidney.

In the cardio-vascular system the tension of the arteries must be noted, for a persistent high blood-pressure will often cause headache, even though there may be no obvious disease of the kidneys.

Valvular lesions of the heart sometimes cause headache, either through anæmia, such as may be associated with aortic regurgitation, or congestion following dilatation of the right ventricle. There is not often much danger of overlooking the cause in either of these conditions, but congestion may, of course, arise from conditions other than those associated with valvular disease.

After the presence of obvious disease of the larger systems has been excluded, the eyes and the sinuses may be thought of, and when thinking of the sinuses it is well to remember that nasal obstruction may be a cause.

Errors of refraction and accommodation, due to astigmatism and hypermetropia, are among the commonest causes of headache, and are often associated with seemingly typical attacks of migraine. Simple myopia does not usually give rise to headaches.

The migraine and toxæmic group of headaches is a large one. The characteristics of migraine are referred to in the next section, and under the hypothesis of toxæmias diet and intestinal action have to be considered, and must be regulated according to the necessities of the individual case.

Headaches sometimes appear to occur reflexly, as the result of sensory impulses through the ears, eyes, and other channels.

Head has shown that many headaches of this kind are "referred" pains depending on diseases of the various viscera.

As already stated, the locality and character of a headache are not usually reliable guides to either the position or the nature of the lesion. Exceptions are, of course, sometimes seen, perhaps chiefly in the case of the headaches of intracranial syphilis, where the painful area is apt to be localised and tender, in addition to which there may be the further characteristics of nocturnal exacerbations.

In most cases of cerebral tumour the headache produced by the general rise of intracranial pressure does not admit of differentiation from that produced at the seat of the growth, though, taken in conjunction with other symptoms, the signs are sometimes very suggestive, as when, for instance, a dull pain occurs over one half of the occipital region and at the same time there is reason to suspect the pressure of a tumour in the cerebellum.

Treatment.—Variations in the blood supply—congestion, hyperæmia, or anæmia—though produced in many different ways, seem to be at the bottom of most headaches, and it is by altering the character of the vaso-motor circulation in some way or other that relief is often obtained.

It goes without saying that, whenever possible, the cause should be removed, but where no definite cause can be found it is necessary to try to ensure the healthy working of all those organs through which the headache is likely to arise, and particularly the eyes, the alimentary canal, and the kidneys. The eyes must be examined and vision corrected, if necessary. The bowels must be kept open by suitable purgatives, and

in the
painful
congestion
is able to
localise
tender
nocturnal
exacerbations

In indefinite cases - make sure the
Eyes, alimentary canal & kidneys
are all in good order.

the kidneys must be washed out by simple diuretics, of which water in plentiful amount is one of the best. At the same time that the output is being looked to, attention must be paid to the intake, and a plain diet, containing a minimum amount of "purin" bodies, prescribed.

For empirical treatment the coal-tar preparations, viz., phenacetin, antipyrin, and antifebrin, are among the most useful drugs. Recently the calcium salts have been used with benefit in cases where headaches seem to be associated with a deficient coagulative power of the blood.^{*} It is said that the subjects who suffer from headaches of this kind are generally of a "lymphatic type," i.e., they have a heavy, listless expression, with pale face and puffy eyelids, and their headaches are generally felt most in the mornings. The lactate of calcium, which is more palatable than the chloride, may be given in doses of 15 to 20 grains three times a day. This drug is said to be also very useful for relieving the morning headaches that follow the taking of too much alcohol on the previous night. Among other drugs that are useful are salicylates, bromides, caffeine, and cannabis indica.

Where there is persistent high arterial tension, vaso-dilators, such as nitro-glycerine and trinitrini, are sometimes given. They have only a transitory effect, as a rule, and more general methods to relieve the pressure should be employed.

Excess of food, by disturbing the chemical processes of digestion, can undoubtedly cause headaches. In the proteid diets it is probably the purins that are harmful, but it has been shown by Hare that headaches of a similar nature can also arise from taking too much carbohydrate food, to which condition he gives the term "hyperpyræmia."

^{*} Dr. George Ross, *Lancet*, Jan. 20, 1906.

MIGRAINE (HEMICRANIA)

Migraine is a paroxysmal neurosis characterised by headache and sickness, which are often preceded by visual symptoms.

Etiology.—More women suffer than men, and there is a strong tendency for the disease to run in families. Occasionally other neuroses, such as hysteria and epilepsy, are prominent in the relatives, but in a large number of instances the patient and his family show no other special form of nervous weakness.

In epileptics the fits sometimes alternate with attacks of migraine, and it is generally supposed that the pathology of migraine is analogous to that of epilepsy, inasmuch as both appear to depend upon a disturbance of the cells of the cerebral cortex; but while it is reasonable to suppose that the greater explosions of epilepsy should sometimes include the lesser of migraine, there is, clinically at any rate, a very wide interval between the average patient with migraine and the sufferer from epilepsy, and patients who are subject to typical attacks of migraine fortunately show no special tendency to become epileptics, though it is undoubtedly easy enough to find plenty of instances where both have occurred and, perhaps, alternated in the same person.

Gout and gouty tendencies have been noted in association with migraine, and it has been considered by some that uric acid is the actual cause of migraine, but of this there is not sufficient proof. In many people the first attacks date from childhood, and appear to become more frequent towards the middle decades of life; in others a few isolated attacks occur from time to time. In those who are the subjects of migraine, attacks are often induced by physical or mental fatigue, indigestion, and work requiring close application of the eyes.

Errors of refraction are notably a cause of the attacks.

General summary of causation.—It is necessary to insist on a broad view being taken of the causation of migraine if success is to be obtained in its treatment. On the whole it appears that certain people are more prone than others to attacks of migraine, and that in them, various causes, such as fatigue, errors of refraction, and dietetic indiscretions, are capable of inducing an attack when they would be harmless to other individuals.

The nature of the condition that determines the tendency to migraine is not definitely known, but it is reasonable to suppose that it is a poison generated either in the alimentary canal or in the tissues themselves during the chemical processes taking place in the latter, and that it acts upon the brain cells either directly or indirectly by the changes produced in the cerebral circulation.

Vasomotor spasm, followed later by dilatation, has always been a plausible hypothesis, and is supported by the pallor of the face and extremities which is often observed at the beginning of an attack, and is followed later by flushing; on the other hand, experiments have not shown the presence of vaso-constrictor nerves in the cerebral vessels.

Symptoms.—The symptoms may be conveniently divided into three groups:

- 1, Ocular.
- 2, Headache.
- 3, Sickness.

1. Ocular symptoms.—In the majority of cases the eye symptoms are the first to occur, but occasionally they are preceded by sensations which pass up a limb, like, but of longer duration than, the aura of epilepsy, and are often accompanied by feelings of giddiness. The visual symptoms take various forms, of which the chief are flashes

of light in front of the eyes, scotoma, coloured spectra, hemianopia, and transient general dimness of vision.

Flashes of bright light may start the attack and then spread out gradually into coloured spectra of zig-zag shape.

The scotoma is usually nearly in the middle of the field of vision, and shows itself to the patient by the existence of a black patch in whatever he looks at, the edges of the patch being zig-zagged, from which the condition has received the names of "fortification spectra" and "teichopsia." The coloured spectra may show themselves as zig-zag ribands of different colours.

Hemianopia is of the simple or homonymous variety, *i.e.* there is a corresponding loss of half vision in each eye, so that the sight is lost towards the right or the left, as the case may be. This often comes on very quickly, and the patient finds he is only able to see one half of the objects he looks at. At other times there is a general dimness of vision, and complete blindness for a time has occasionally been observed.

2. *Headache*.—Following on the eye symptoms comes the headache. This often begins at one spot in the region of the right or left temple, and thence spreads over one half (hemicrania) or the whole of the head. At other times the painful spot on the one side is followed by a pain over the corresponding area of the other side before the headache becomes general.

3. *Sickness*.—The headache generally increases in intensity for a time, and is succeeded by a sensation of nausea, which may be followed by retching and vomiting; the headache, in its turn, fades away, and so the attack passes off.

Additional sensory symptoms which sometimes occur are numbness and tingling or even temporary weakness of a limb, and occasionally a passing aphasia may be met with.

Duration.—The duration of an attack varies from a few hours to a day or more. Some people are incapacitated for about two days, but, as a rule, recovery takes place after a night's rest.

Prognosis and treatment.—Cases in which a tendency to the attacks is only slight can often be cured by getting rid of some form of irritation which excites the attacks. It is in this way that many patients get relief by wearing suitable glasses.

In some, the most careful correction of errors of refraction and the removal of any other obvious source of irritation make but little difference, and in others, again, no error of refraction of any importance exists.

Diet.—The diet should be light, and, although there is no proof that uric acid is the cause of the attacks, many cases certainly seem to benefit by abstaining from meat in any quantity.

In severe cases it is worth while to go further and try what a strict purin-free diet will do.

Early hours should be kept, fatigue should be avoided, and the bowels kept regular.

For warding off attacks, bromides and salicylates are among the most useful drugs, and act better in combination than when given separately. When the patients generally feel the symptoms on first waking, the medicine should be taken the last thing at night. In other cases it may be given twice or three times a day for a time, and then gradually reduced.

Very little can be done to cut short an attack beyond resting and applying cold to the head.

Such drugs as phenacetin, antipyrin, and caffeine, if taken at the very beginning, sometimes afford relief, but once the symptoms have gained a fair hold drugs seem to have little effect; probably the stomach is not in a suitable condition to absorb them.

The methods of treating migraine may be summed up as follows:

BETWEEN ATTACKS.

1. Avoidance of undue fatigue and excitement.
2. Correction of errors of refraction or any other source of peripheral irritation.
3. Regulation of diet. Digestible food, with restriction of meat.
4. Prescription: e.g., potassii bromidi (gr. x. to xv.), sodii salicylatis (gr. x. to xv.), spiritus chloroformi (m. xx.), aq. ad ̄ i.—taken three times a day, or a larger dose every night.

DURING ATTACKS.

1. Rest in a darkened room.
2. Cold to head.
3. Phenacetin (gr. x.), antipyrin (gr. x.), or antifebrin (gr. iii.).

OPHTHALMOPLEGIC MIGRAINE

This is a somewhat uncommon condition, in which the periodic headaches are accompanied by temporary paralysis of the third nerve with its attendant symptoms of ptosis, strabismus, and fixed, dilated pupil.

R
 1. Potassii Bromid. gr. x—
 Sodii Salicylatis gr. x—
 Spiritus Chloroformi (m) xx
 Aquam ad. unciam.
 Fiat histuram —
 bulk - 3 iv
 Lc 3 i

CHAPTER XLVIII

VERTIGO

Etiology. — A sensation of giddiness may accompany a great many morbid conditions, of which the most important are disorders of the ears, eyes, heart, stomach, and kidneys; intracranial growths, especially those connected with the cerebellum; and epilepsy. Alcoholism, excessive smoking, and other toxæmic conditions may also cause vertigo.

Excluding the vertigo of epilepsy, the most important and the most common causes of giddiness are disorders of the ear. Before the frequency of aural vertigo was realised the cause was, in the majority of cases, attributed to gastric trouble, but it is now generally acknowledged that vertigo arising from indigestion is, as a rule, of comparatively slight importance.

AURAL VERTIGO

So far as the ear is concerned, the sense of equilibrium depends on the pressure of the endolymph and on the state of the nerve terminals in the semicircular canals, from which there is direct communication with the cerebellum. Anything, therefore, that varies the pressure or alters the irritability of the nerves may upset the normal equilibrium and cause vertigo.

The pressure of the endolymph within the semicircular canals may be modified by chronic inflammatory changes in the middle ear, either of gouty, senile, or syphilitic origin, whereby the

intensity of the vibrations conducted through the chain of ossicles is increased. Wax, which is one of the commonest causes of vertigo, probably produces its effects in the same way, for a solid piece, if pressed against the drum of the ear, serves as an additional conductor, and causes the intensity of the vibrations reaching the labyrinthine nerves to be increased. In other instances an undue irritability of the nerve fibres or their centres is the cause of the vertigo. The labyrinth itself may be the seat of injuries, of effusions, or of inflammations.

The symptoms of aural vertigo are also sometimes associated with high arterial tension and Bright's disease, quite apart from the giddiness which may be a symptom of uræmia.

Vertigo of labyrinthine origin is known as Menière's disease, but the term is often used to include other forms of aural vertigo.

Symptoms.—In a typical case of aural vertigo the patient suddenly feels giddy, and either has a sensation of movement or actually moves; at other times, the movement of surrounding objects is the chief thing noticed. In severe cases the patient may suddenly be thrown to the ground or may only save himself by holding on to some neighbouring support.

After the giddiness has lasted a variable time it is often succeeded by nausea and vomiting. Occasionally temporary diplopia and nystagmus have been noticed (Gowers). There may be slighter attacks between those of greater severity.

Tinnitus often immediately precedes a paroxysm and sometimes also persists between the attacks. Some degree of deafness is also frequently to be found on one or other side. The nature of the deafness can be tested by a tuning-fork. The vibrations from a tuning-fork placed on the forehead are ordinarily heard in both ears equally, but if the conducting apparatus of the

ear is impaired they are heard loudest on the side of the deaf ear.

On the other hand, when the deafness is of nerve origin the vibrations of the tuning-fork placed on the forehead are heard best on the sound side.

Further, in nerve deafness, after the vibrations of the tuning-fork placed on the mastoid have been lost they can generally still be heard when the instrument is held opposite the meatus.

Diagnosis.—Attacks of *vertigo* associated with *deafness* and *tinnitus* are the main points to be established.

The vertigo of *epilepsy* is sometimes difficult to differentiate, but if the attacks are repeated frequently, some positive signs of epilepsy, in the shape of loss of consciousness, convulsions, biting the tongue, and involuntary micturition, are almost sure to be obtained. The most difficult cases to decide are those in which the patient actually falls, for in those circumstances there is naturally some confusion of mind which is not easy to differentiate from the loss of senses due to epilepsy. If a history of loss of consciousness can be established, the condition is almost certainly one of epilepsy. It is doubtful if vertigo of anything like the severity described above ever arises from *gastric disorders*, though, of course, "dizziness" and "giddiness" are often complained of by patients who suffer from indigestion.

Tumours of the brain, especially those of the cerebellum, are frequently accompanied by giddiness, but other signs of intracranial pressure are generally present.

It is necessary to remember that occasionally the giddiness of *hysterical patients* may simulate that of Menière's disease, but, needless to say, it is necessary to be very cautious before ascribing a case to this cause.

Prognosis.—In aural vertigo only very rarely

is such an acute attack as Menière recorded associated with changes likely to endanger life. Possibilities of improvement must, of course, largely depend on the anatomical changes that have occurred. In a large number of cases partial, if not entire, relief is obtained.

Treatment.—The ear must be carefully examined, and if any wax is present it must be removed. In some cases this alone is sufficient to effect a cure. If the giddiness cannot be accounted for in this way, recourse must be had to drugs, and of these the bromides, in 15- to 20-grain doses twice or three times a day, are probably the most efficacious. Rheumatic and gouty cases may derive benefit from salicylates and iodides, which may be given in combination with the bromides. Quinine sulphate is a drug that was recommended by Charcot, who considered it necessary to give it in large doses, but many authors consider small doses to be equally efficacious. Counter-irritation in the shape of a blister over the mastoid is sometimes useful. If these measures bring no definite relief, it may be well to put the patient to bed for a week or two, for complete rest is often a most useful therapeutic measure. If the arterial tension is too high, purgatives and careful dieting are often beneficial. If all else fails and the attacks continue to be unbearable, the question of obliterating the semi-circular canals may be entertained.

Treatment of vertigo from other causes.—All forms of vertigo are generally relieved to some extent by taking bromides, and any further course of treatment must depend upon the cause, whether it be heart, stomach, arterial degeneration, cerebral tumours, or ocular palsies.

CHAPTER XLIX

VASOMOTOR NEUROSES

DISORDERED functions of the vasomotor nerves are sometimes the cause of various unpleasant symptoms, of which most can be grouped under one of the following headings:

1, Raynaud's disease; 2, angio-neurotic œdema; 3, erythromelalgia; and 4, acroparæsthesia.

1. **Raynaud's disease.**—The **symptoms** of Raynaud's disease can be divided into three stages:

(1) Local "syncope," in which the fingers, toes, and perhaps the nose and ears, become pale and "dead."

(2) Local "asphyria," in which the initial pallor is followed by congestion, which gives the part a blue colour.

(3) Gangrene, in which small parts of the asphyxiated areas necrose and separate, leaving little scars.

A large number of patients whose symptoms come under this heading never experience all these three stages. Many people habitually suffer from "dead" fingers, especially in cold weather or after putting the hands into cold water, and in some of these the stage of "local" asphyxia is also experienced; necrosis occurs in only comparatively few.

Spasms of the vessels of the extremities appear to be the cause of the disease, and the spasm is usually attributed to a "vasomotor neurosis."

What causes the neurosis is uncertain, but probably, at any rate in some cases, it is the result of a toxæmia. Symptoms of a similar nature to those described above may be found associated with syringo-myelia, and they are also especially prevalent during the early stages of some cases of rheumatoid arthritis.

Vasomotor affections of the kidney resulting in paroxysmal hæmoglobinuria are sometimes associated with Raynaud's disease, and closely allied to it clinically are the ordinary chilblains of winter weather.

Friction and electricity are the most useful forms of treatment, and in the case of the latter the constant current, applied daily, is the most efficacious method of administering it. The hand and the positive pole should be placed in a bowl of warm salt and water, and the negative pole should be applied to the upper part of the limb.

Drugs are of little direct value, but those that have a general tonic action may be given, and some beneficial results are sometimes obtained from nitro-glycerine, sodium nitrite, and other vasodilators.

The symptoms occur in paroxysms and are always worse in cold weather; hence care should be taken to wear warm gloves and socks.

2. Angio-neurotic œdema.—Œdema occurs in the skin and sometimes in the mucous membranes of different areas of the body. At one time the face may be the seat of the swelling, and at another the limbs or the trunk. The œdema lasts a variable time and then disappears. It gives rise to some discomfort and irritation, and if present in the mouth and pharynx may make swallowing uncomfortable; œdema of the glottis may occur and give rise to symptoms that are dangerous to life.

Closely allied to it clinically are the urticarias

which follow the ingestion of shell-fish, pork, and other foods.

The history of the varying nature of the swelling generally helps in distinguishing its nature, but the urine should always be carefully examined so as to exclude the possibility of Bright's disease being the cause of the œdema.

Sometimes more than one member of a family suffers from angio-neurotic œdema.

Treatment.—It is not easy to cure angio-neurotic œdema. Attention should be directed towards the alimentary canal, and careful dieting, with abstention from the class of foods that usually give rise to urticaria, may be tried. With the same object in view, intestinal antiseptics may be given.

It is important to maintain a high standard of general health.

3. **Erythromelalgia.**—This condition usually occurs in the distal parts of the arms or legs. The arteries dilate, the skin becomes hot and red, and there is great pain. After a time the redness of the skin disappears, and is replaced by a bluish colour. The intensity of the symptoms is increased by holding the affected limb in a dependent position, and also by warmth or exercise. When arterial degeneration is present, intermittent claudication and cramps may be associated with this disease.

Symptoms of erythromelalgia may be met with in association with various organic diseases of the nervous system, *e.g.*, multiple neuritis and tabes, and with arterio-sclerosis; at other times no sign of organic changes can be found, and the disease is then attributed to a vasomotor neurosis. The symptoms are generally relieved by keeping the limb high and by cool applications to the affected part. Massage and electricity may also be useful, as they are in Raynaud's disease.

4. **Acroparaesthesia of vasomotor origin.**—

Here the patient complains of tinglings, pins and needles, and numbness of the extremities (usually of the arms). In most of the cases the symptoms come on in the night. The patient goes to bed and falls asleep, but after a time wakes up with tingling and numbness, which are sufficiently severe to prevent any more sleep being obtained. On getting up and using the limbs the sensations usually pass off, but they are very apt to recur during the day if the patient rests for any length of time, especially if near a fire so that the limbs get hot.

The symptoms are more commonly found in women than in men, and especially in those who are engaged a good deal in washing. The use of alcohol also seems to favour their onset.

The women sufferers are often overworked and generally debilitated, and some of them are at the menopause period of life.

Tingling, numbness, and "crawling sensations," accompanied in some instances by some anæsthesia, are frequently met with over the area of the thigh, which derives its cutaneous nerve-supply from the external cutaneous, and to this condition the term meralgia paræsthetica has been applied. In the majority of cases no lesion of the nerve can be found, and we have to fall back on a vasomotor neurosis as the cause.

Diagnosis. — Before concluding that the symptoms of acroparæsthesia are due to a functional disturbance, care must be taken to exclude the presence of peripheral neuritis, locomotor ataxy, or other organic conditions with which symptoms of a similar nature may be associated.

The **prognosis** of these cases is, on the whole, good, if the patient can be placed amidst favourable surroundings, but recovery is generally slow and the relapses are frequent.

Treatment. — Iron, nux vomica, and other tonics are helpful, with a view of restoring the

health, but a thorough change is best of all if it can be procured.

Electricity and massage are both useful.

It is difficult to give anything which will be certain to relieve the symptoms at the time, but the bromides seem to afford as much relief as anything. If sleep be much interfered with, other hypnotics may be required.

CHAPTER I

EXOPHTHALMIC GOITRE (GRAVES'S DISEASE, BASEDOW'S DISEASE)

Etiology. — Exophthalmic goitre is far more commonly seen in women than in men. In the majority of the cases the symptoms first appear between the ages of fifteen and forty, and it is rare to find the disease arising in elderly people.

In many instances nothing that seems to have definitely led up to the origin of the disease can be traced, but in a certain proportion of the cases fright, shock, and long-continued strain of the nervous system appear to bear a decided relation to the onset of the symptoms.

Pathology. — The view that exophthalmic goitre arises from lesions of the cervical sympathetic or of the central nervous system now receives less support than formerly, and the hypothesis that meets with most favour at the present day is that which attributes the symptoms to a toxæmia arising from a modification in the secretion of the thyroid or parathyroid glands. The influence which the secretion of the thyroid gland normally exerts on the general metabolism of the body can be fully appreciated from the ill effects, so well seen in myxœdema, which arise on its removal, and if disappearance of the gland can produce such far-reaching results it is not unreasonable to presume that modifications in the composition or amount of the secretion might produce peculiar effects of another nature. This view receives some support from the constancy

with which changes in the thyroid gland are found in exophthalmic goitre, and it is also a noticeable feature that many of the symptoms are almost the exact opposite of those which occur in myxœdema, where the secretion of the gland has disappeared. Moreover, excessive doses of thyroid gland are liable to cause symptoms which in many respects are not unlike some of those that are commonly associated with exophthalmic goitre; and, lastly, relief from the symptoms of exophthalmic goitre has occasionally been obtained by partial extirpation of the gland.

In some instances an enlarged thymus gland has been found, and changes of inconstant and somewhat indefinite character have been noted in the ganglia of the cervical sympathetic and in the medulla.

Symptoms.—The onset in most cases is gradual; only occasionally is it rapid.

The order in which the signs appear differs, and there is also great variation in their number and intensity, but the principal ones are four, viz.: Enlargement of the thyroid gland, tachycardia, exophthalmos, and tremors.

Enlargement of the thyroid gland.—This sign is present at some period or another in nearly all cases, and it may precede other symptoms by a long time. Like the other manifestations of this disease, the enlargement is not only very various in different cases, but is apt to vary from time to time in the same case. In some patients it is so slight as to be almost unnoticeable, while in others the gland is symmetrically or asymmetrically enlarged to a great size. The vascularity of the enlarged gland is usually increased, the pulsation of the larger arteries running into it can be seen and felt, and not unfrequently a thrill and a murmur over the tumour can also be detected.

Tachycardia.—Rapidity of the pulse-rate is a characteristic symptom; from 100 to 120 beats per

minute are commonly met with, and frequently the number is very much higher.

Pulsation of the heart is generally more obvious than usual, and a systolic murmur can often be heard at the apex.

Exophthalmos.—The prominence of the eyeballs gives the striking appearance of fright to the face. Protrusion of one eye before the other sometimes occurs.

The cause of the exophthalmos has been much debated. It has been attributed to various conditions, *e.g.*, increased vascularity, increase of fatty tissue at the back of the eyeball, and contraction of Müller's unstriated muscle, which runs across the back of the orbit.

Other ocular symptoms which can frequently be observed are: Von Graefe's sign, in which the dropping of the eyelids is delayed when the eyes are directed downwards; Stellwag's sign, in which there is retraction of the upper lid due to spasm of the unstriated muscle fibres; and a difficulty in converging the eyes to view near objects. (*more* *mobile*)

Tremors.—A fine rhythmical tremor of the hands, which can often be better felt than seen, is met with in most of the cases.

The remainder of the symptoms, which are numerous, may be briefly summarised under the headings of the different systems to which they are attached.

Nervous system.—Patients suffering with exophthalmic goitre are apt to be nervous, excitable, and very easily upset. They often sleep badly, and they may develop symptoms of hysteria and other slight forms of mental derangement. In a few cases the mental changes are profound enough to constitute a state of insanity.

Respiratory system.—In the more severe cases attacks of dyspnœa may occur.

Alimentary system.—The appetite is variable, as also is the power of digestion. Vomiting and

diarrhœa, more commonly the latter, are both met with.

Renal system.—The urine is generally plentiful in amount, and often contains a high percentage of urea. Glycosuria and traces of albumen are found in some cases.

The skin.—The skin is usually moist, and perspiration is easily induced. Brown pigmentation is frequently noticed.

The general nutrition is poor, and the patients are frequently emaciated. In women uterine disturbances are often met with.

Diagnosis.—There is no difficulty in diagnosing a case in which the main symptoms are well developed. There is, however, always a possibility of overlooking the nature of the disease when general symptoms, such as nervousness and loss of weight, are the most prominent features.

Prognosis.—Improvement, which is usually a very slow process, occurs in a considerable number of cases. In some the symptoms entirely subside, but in the majority some traces of exophthalmos still remain, as also frequently do the nervous and excitable temperaments.

A fatal termination from the direct effects of the disease is distinctly uncommon, but it occasionally happens that the disease runs an acute course, and, the patient becoming delirious, death takes place with all the symptoms of profound toxæmia. In a few cases, where the functions of the thyroid gland are eventually destroyed, the symptoms of myxœdema have supervened.

Treatment.—Among the drugs which are of most service, arsenic, belladonna, bromides, iron, and digitalis may be mentioned. Arsenic, chiefly in the form of liquor arsenicalis, is one of the most popular for general use, but bromides and belladonna are both serviceable on account of their sedative effects. Iron acts as a general tonic, and digitalis is frequently useful for steadying the

heart. When the enlargement of the thyroid is accompanied by much pulsation, comfort is often obtained by placing an ice-bag on it.]]

Applications of electricity over the course of the cervical sympathetic and over the thyroid gland sometimes seem to produce beneficial effects. The interrupted and constant currents may both be used.

Preparations of the various glands, *e.g.*, thyroid, thymus, and suprarenal, have been tried, but with no distinctive good results. One would, indeed, expect preparations of thyroid gland to be harmful if the hypothesis which ascribes the disease to hypersecretion be correct, but, as a matter of fact, cases have been recorded in which benefit appears to have been obtained from their use. Such results are not necessarily to be taken as invalidating the hypothesis of disordered action of the thyroid gland, for it may be that in some cases the modification of the secretion is one of quality rather than of quantity.

Administration of preparations of the thymus gland has also sometimes appeared to be followed by relief of symptoms, but there is no general consensus of opinion in favour of the habitual use of this class of drug.

It is very important to recommend such measures as are likely to improve the general health. Rest, sometimes under the Weir Mitchell system, is important, as also are quiet surroundings and general tranquillity of mind.

Operations for partial removal of the thyroid are sometimes undertaken, but the risks which this procedure entails only justify its use in exceptional cases.) ?

CHAPTER LI

TETANUS (TRISMUS, LOCKJAW); HYDROPHOBIA

TETANUS

Etiology.—Tetanus is an acute infective disease resulting from the entrance into the body of the tetanus bacillus.

The tetanus bacillus, which is of a drumstick shape, flourishes chiefly in the soil, and in most cases is introduced into the body through punctured and lacerated wounds. Infants are liable to be infected through the umbilical wound (tetanus neonatorum), and parturient women through the placental surface (puerperal tetanus).

Pathology.—The tetanus bacilli remain in the neighbourhood of the wound, and cause symptoms by diffusion of the toxins to which they give rise. These toxins have a special affinity for nervous tissue, by which they appear to be “fixed” after the manner of certain stains.

After death, minute hæmorrhages can be seen in different parts of the central nervous system, which throughout is found to be intensely congested.

Definite changes in the cell bodies of the brain and cord can often be distinguished, the most characteristic of which are swelling of the cell bodies and modifications of the staining properties of the chromatophile granules.*

Symptoms.—After the patient has become

* Alexander G. R. Foulerton and Campbell Thomson, *Trans. Roy. Med. Chir. Society*, vol. lxxxiii., p. 900.

infected there is a period varying in duration from two to three days to as many weeks before the symptoms appear. The first intimation of anything wrong is generally a sensation of stiffness in the muscles of the neck, which is soon followed by spasms in the muscles of the jaw and face, by which the jaw becomes locked and the features distorted to produce the "risus sardonicus." Superadded to these tonic contractions are spasms of the muscles of the trunk and limbs, which occur with great frequency and with such intensity as to cause opisthotonos and other contortions of the body. The slightest peripheral stimulation, such as from a loud noise, a draught of air, or an attempt to move, is apt to bring on a spasm.

Contractions of the respiratory and laryngeal muscles give rise to great distress and difficulty in breathing, and pain produced by the generalised cramps is also a prominent feature.

The frequency of the spasms depends upon the intensity of the infection. The temperature may be raised slightly or not at all during the earlier stages of the disease, but there may be hyperpyrexia towards the end. The mind usually remains clear, and death takes place from exhaustion and failure of respiration.

Diagnosis.—The mode of onset, characterised by rigidity of the muscles of the neck, jaw, and face, when following a traceable cause of infection, seldom leaves room for much doubt concerning the nature of the disease.

Hydrophobia is the disease in which the symptoms most closely resemble those of tetanus, but in hydrophobia the "lockjaw" is absent, and the spasms are especially concentrated on the muscles connected with swallowing, so that attempts to drink are at once followed by an attack. There is in hydrophobia also the likelihood of a history of the possibility of infection having occurred from a bite of a dog or some other animal.

Poisoning from *strychnine* gives rise to spasms which resemble those of tetanus, but there is more complete flaccidity of the muscles in the intervals between the spasms, and trismus is absent. Moreover, the onset is likely to be far more sudden.

In *tetany* the distribution and characters of the contractions in the hands and feet usually obviate any possibility of confusion even when the spasms invade the muscles of the trunk.

Prognosis.—A patient who is infected by tetanus bacilli is always in a dangerous position, and the mortality is said to be more than 80 per cent.

The longer the incubation period the better is the outlook; and when the spasms have set in, the intensity of the infection and the gravity of the case can be to some extent gauged by the strength and frequency with which the convulsions occur.

Treatment.—The site at which infection has taken place should be cleaned and disinfected. This may influence the course of the disease, but, as already stated, the symptoms appear to be due to the diffusion of toxins, and therefore they, unfortunately, do not subside with the removal of the bacilli.

The patient should be isolated in a darkened room, and should as far as possible be protected from all forms of stimulation which are liable to intensify the strength of the spasms.

Drugs must be used to produce sedative effects, and for this purpose bromides, morphia, cannabis indica, and chloral will naturally suggest themselves. When the convulsive attacks are very severe, inhalation of chloroform may be followed by some relief.

Nutrition must be maintained, and if necessary the patient must be fed by a nasal tube.

Periods of dyspnoea may be tided over by performance of artificial respiration.

Antitoxins can be procured, and should be

injected, preferably into the brain tissue itself; but, up to the present, beneficial effect from their use cannot be predicted with any certainty.

HYDROPHOBIA (RABIES)

Etiology.—Hydrophobia is transmitted to man by the saliva of animals, such as dogs, cats, and wolves, which are at the time suffering from rabies.

Pathology.—After death the principal alterations found in the central nervous system are hyperæmia, minute hæmorrhages, and extravasation of leucocytes into the perivascular tissues.

Symptoms. The incubation period is very variable, but the usual interval which precedes the onset of symptoms is about six weeks, though cases have occasionally been recorded in which twelve or even eighteen months had elapsed before the signs of infection made their appearance.

Vague sensations referred to the seat of the wound, which in the meantime may have healed perfectly, often first lead to an idea that something is wrong, and this suspicion is soon confirmed by the advent of convulsive attacks somewhat similar to those of tetanus, but differing from them in the fact that the intensity of the spasms is especially concentrated on the muscles concerned in swallowing. It is due to this latter fact that the act of drinking becomes so difficult, for violent spasms occur whenever the patient attempts to take fluid, and even the sight of water is often sufficient to bring them on.

These convulsive seizures soon spread to other parts and are brought on by the slightest stimulation, such as a sudden noise or a draught of air impinging on the body; and when the muscles of respiration are implicated there occur intense and painful attacks of dyspnœa, which then become a prominent feature of the case.

The temperature is raised, and wandering of the mind with hallucinations and delusions is frequently present.

Death occurs from exhaustion brought about by the spasms, and it may be preceded by a paralysis beginning in the lower limbs, similar to that which occurs in animals.

Diagnosis. — The main differences between the symptoms of hydrophobia and tetanus have already been mentioned (p. 463).

The convulsions arising from poisoning by strychnine are not concentrated in the muscles concerned in swallowing, as they are in hydrophobia. Tetany can scarcely give rise to any serious difficulty, but hysteria has occasionally been known to simulate the symptoms of hydrophobia.

Prognosis. — The danger of a hydrophobia occurring after the bite of an animal suffering from rabies is greater when the patient is bitten on unprotected parts of the body, such as the face and hands, than when the animal's teeth have passed through the clothes, the latter presumably acting as a barrier to the entrance of the poison. About 15 per cent. of the patients bitten by dogs suffering from rabies eventually develop symptoms of hydrophobia.

Treatment. — Whenever hydrophobia shows itself it should be promptly dealt with by strictly enforcing the muzzling of dogs. A recent bite should be freely cauterised as soon as possible, and if none of the usual methods of doing this are at hand, rough-and-ready means, such as the application of some heated instrument to the wound, can usually be devised. Diffusion of the poison may be also retarded by applying a ligature tightly round the limb and encouraging free bleeding.

Once the poison has been absorbed, the only method of treatment which can be relied on to stave off the disease is that of inoculation of the attenuated virus as discovered by Pasteur. The

length of the incubation period makes it possible to render the patient immune by the inoculation of attenuated virus of gradually increasing strength, which is prepared from the spinal cords of rabbits that have been infected with the disease. By this means the mortality, which in untreated cases is very high, has been greatly reduced, and, provided the case is taken in time and treated thoroughly, there is every chance of the toxin being neutralised before it has had time to cause any symptoms.

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